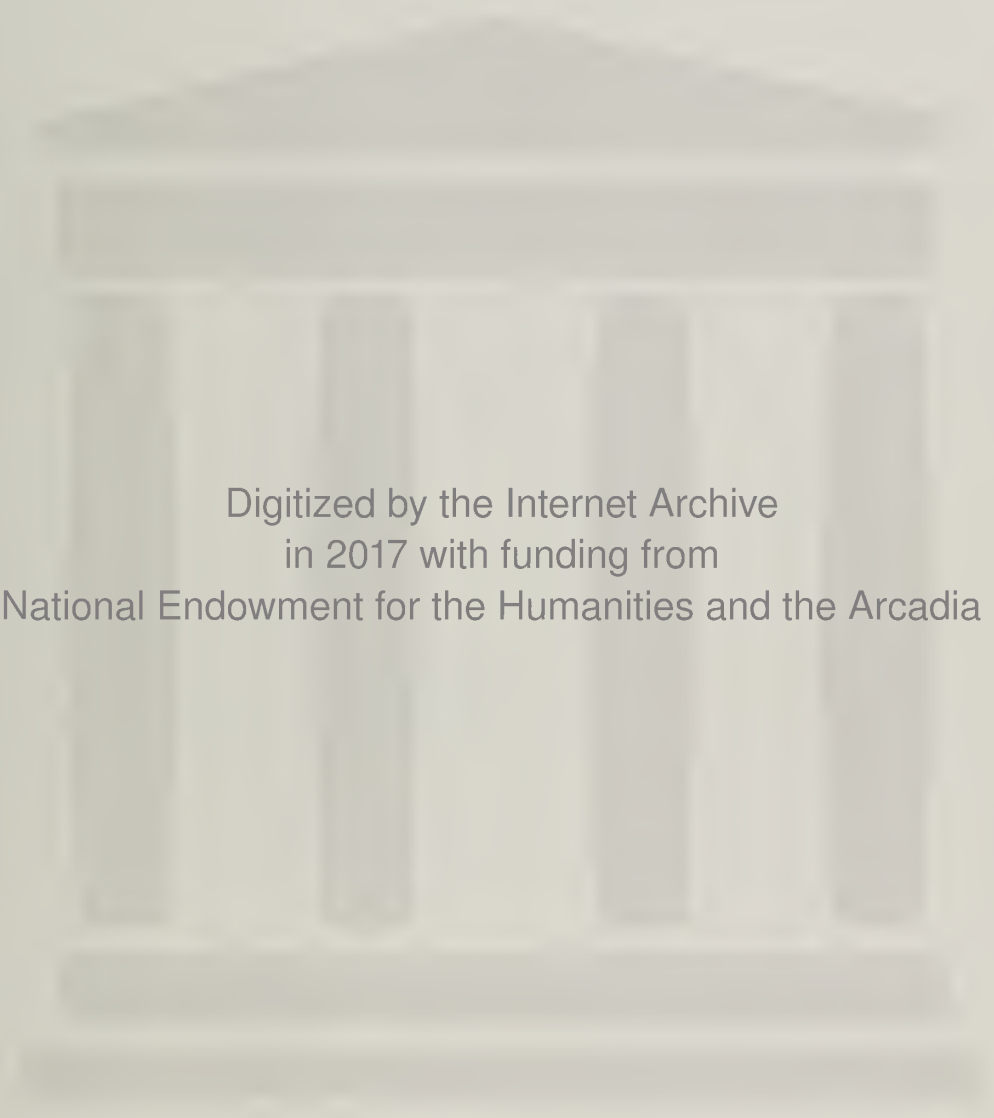
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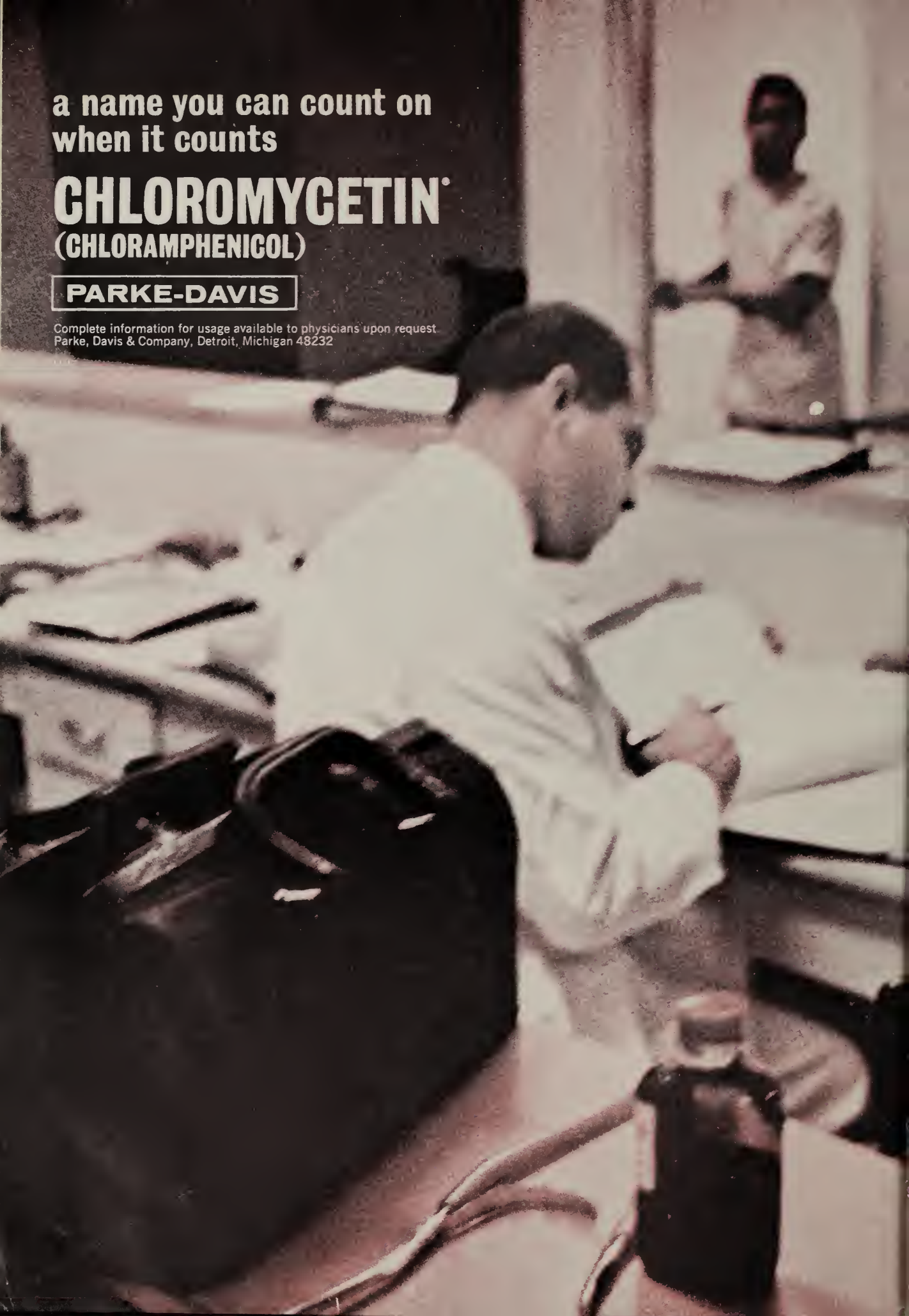
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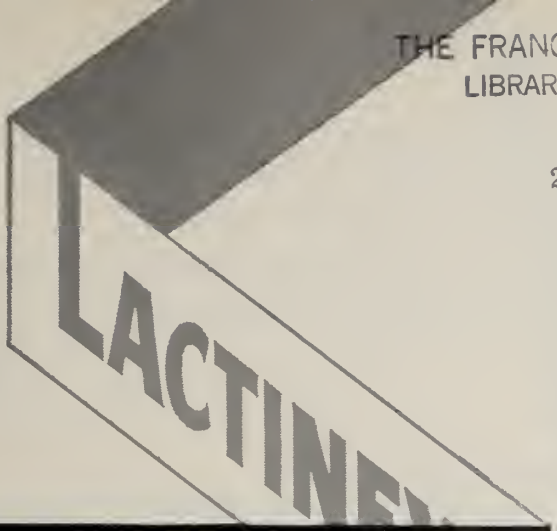
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when it counts
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LACTINEX®

TABLETS & GRANULES

■ **to help restore and stabilize the intestinal flora**

■ **for fever blisters and canker sores of herpetic origin**

LACTINEX contains both *Lactobacillus acidophilus* and *L. bulgaricus* in a standardized viable culture, with the naturally occurring metabolic products produced by these organisms.

First introduced to help restore the flora of the intestinal tract in infants and adults,[1,2,3,4] LACTINEX has also been shown to be useful in the treatment of fever blisters and canker sores of herpetic origin.[5,6,7,8]

No untoward side effects have been reported to date.

Literature on indications and dosage available on request.

References:

(1) Siver, R. H.: CMD, *21*:109, September 1954. (2) Frykman, H. H.: Minn. Med., *38*:19-27, January 1955. (3) McGivney, J.: Tex. State Jour. Med., *51*:16-18, January 1955. (4) Quehl, T. M.: Jour. of Florida Acad. Gen. Prac., *15*:15-16, October 1965. (5) Weekes, D. J.: N.Y. State Jour. Med., *58*:2672-2673, August 1958. (6) Weekes, D. J.: EENT Digest, *25*:47-59, December 1963. (7) Abbott, P. L.: Jour. Oral Surg., Anes., & Hosp. Dental Serv., 310-312, July 1961. (8) Rapoport, L. and Levine, W. I.: Oral Surg., Oral Med. & Oral Path., *20*:591-593, November 1965.

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b.i.d.

The sensible schedule that covers the patient day and night

If your objective in the use of a broad-spectrum antibiotic is prolonged action, with high blood levels, then you know why b.i.d. DECLOMYCIN is considered to be a sensible dosage schedule.

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The b.i.d. dosage of DECLOMYCIN gives you the comfortable assurance that the patient is well-covered, *day and night.*

In clinical practice, blood levels produced by a therapeutic dose of DECLOMYCIN are high, prolonged, and effective; because of high serum binding and slow renal clearance. And if there's a broader susceptibility pattern of organisms, we've yet to see it.

There is no need to give higher daily dosage than 300 mg b.i.d., except in venereal diseases and Eaton Agent pneumonia.

DECLOMYCIN®
DEMETHYLCHLORTETRACYCLINE

Lederle

Prescribing information on next page.

b.i.d. The sensible schedule that covers the patient day and night

DECLOMYCIN Demethylchlortetracycline should be equally or more effective therapeutically than other tetracyclines when the offending organisms are tetracycline-sensitive.

Contraindication: History of hypersensitivity to demethylchlortetracycline.

Warning—In renal impairment, usual doses may lead to excessive accumulation and liver toxicity. Under such conditions, lower than usual doses are indicated, and, if therapy is prolonged, serum level determinations may be advisable. A photodynamic reaction to natural or artificial sunlight has been observed. Small amounts of drug and short exposure may produce an exaggerated sunburn reaction which may range from erythema to severe skin manifestations. In a smaller proportion, photoallergic reactions have been reported. Patients should avoid direct exposure to sunlight and discontinue drug at the first evidence of skin discomfort. Necessary subsequent courses of treatment with tetracyclines should be carefully observed.

Precautions—Overgrowth of nonsusceptible organisms may occure. Constant observation is essential. If new infections appear, appropriate measures should be taken. In infants, increased intracranial pressure with bulging fontanels has been observed. All signs and symptoms have disappeared rapidly upon cessation of treatment.

Side Effects—Gastrointestinal system—anorexia, nausea, vomiting, diarrhea, stomatitis, glossitis, enterocolitis, pruritus ani. Skin—maculopapular and erythematous rashes. A rare case of exfoliative dermatitis has been reported. Photosensitivity; onycholysis and discoloration of the nails (rare). Kidney—rise in BUN, apparently dose related. Transient increase in urinary output, sometimes accompanied by thirst (rare). Hypersensitivity reactions—urticaria, angioneurotic edema, anaphylaxis. Teeth— dental staining (yellow-brown) in children of mothers given this drug during the latter half of pregnancy, and in children given the drug during the neonatal period, infancy and early childhood. Enamel hypoplasia has been seen in a few children. If adverse reaction or idiosyncrasy occurs discontinue medication and institute appropriate therapy.

Average Adult Daily Dosage: 150 mg q.i.d. or 300 mg b.i.d. Should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products. Treatment of streptococcal infections should continue for 10 days, even though symptoms have subsided.

In the treatment of syphilis a dosage schedule of a total of 12 to 18 Gm. given in equally divided doses over a period of 10 to 15 days should be followed. Close follow-up observation of the patient is recommended, including appropriate laboratory tests, since demethylchlortetracycline has not had adequate evaluation in all stages of syphilis. Spinal fluid examination should be included as part of this follow-up.

Acute gonococcal anterior urethritis in males has been treated effectively with a single dose of 600-900 mg. of DECLOMYCIN Demethylchlortetracycline. Individuals unable to tolerate large single doses due to gastrointestinal side effects may be treated with 150 mg. every 6 hours for a minimum of 4 doses or 300 mg. every 12 hours for a minimum of 2 doses. Females should be treated with a dosage of 150 mg. every 6 hours or 300 mg. every 12 hours until a cure is effected.

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A tablet-a-day provides:

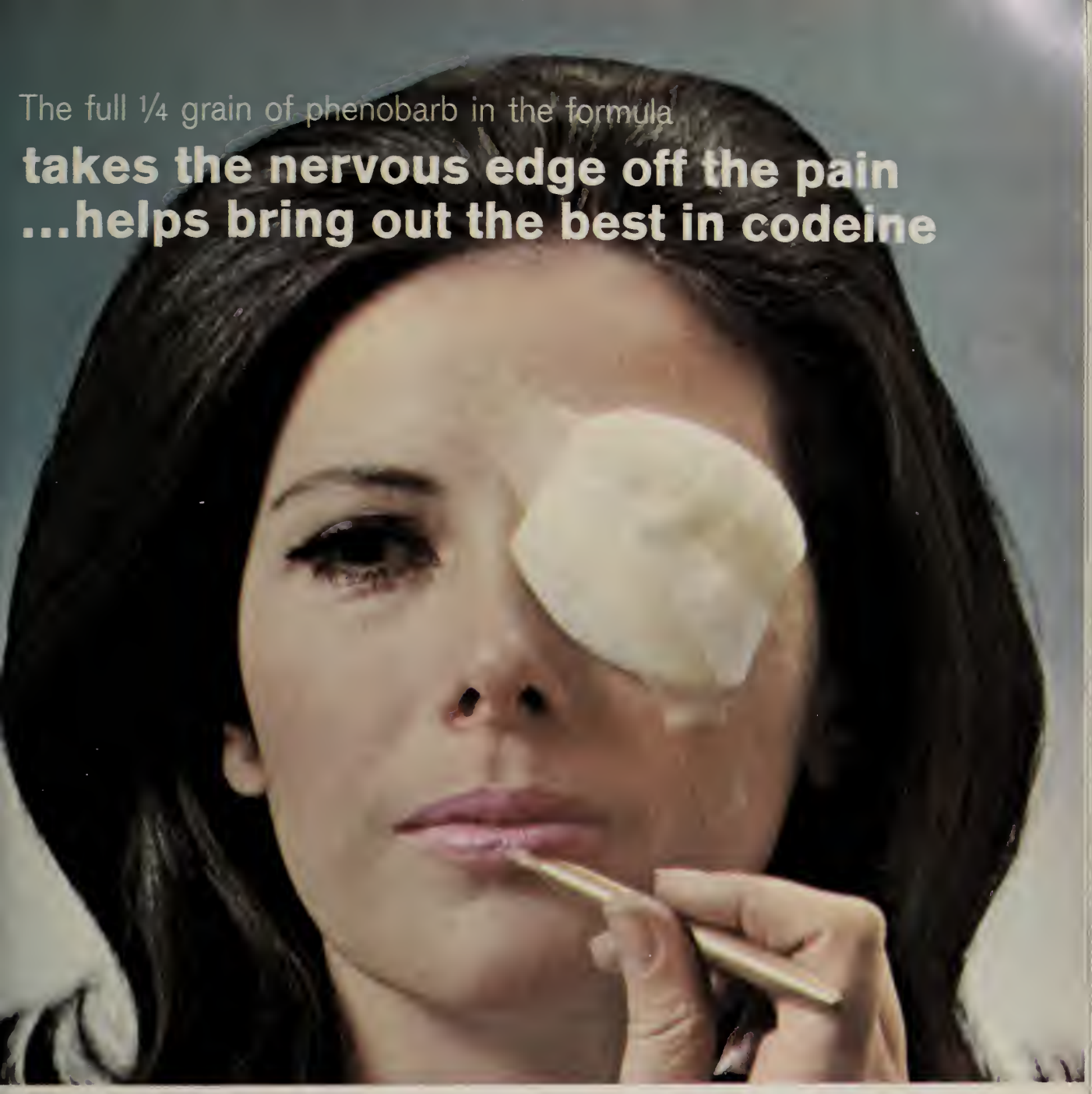
● Elemental Iron (as Ferrous Fumarate)	100 mg
● Dioctyl Sodium Sulfosuccinate (to counteract constipating effect of iron)	100 mg
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Vitamin B_2	7.5 mg
Vitamin B_6	7.5 mg
Vitamin B_{12}	50 mcgm
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Niacinamide	30 mg
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Pantothenic Acid	15 mg

Bottles of 60

* anticostive, *adj.* (*anti* opposed to + *costive* causing constipation.) Against constipation. (Now isn't that a good idea in an iron-containing hematinic? We'll send you samples if you'll send a request on your Rx blank, addressed to Department 150.)

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takes the nervous edge off the pain ...helps bring out the best in codeine

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Each capsule contains:

Phenobarbital (¼ gr.) ..... 16.2 mg.
(Warning: may be habit forming)
Aspirin (2½ gr.) .....162.0 mg.
Phenacetin (3 gr.) .....194.0 mg.
Hyoscyamine sulfate .....0.031 mg.
Codeine phosphate.....¼ gr. (No. 2), ½ gr. (No. 3), 1 gr. (No. 4)
(Warning: may be habit forming)

Contraindications: Hypersensitivity to any ingredient.

Precautions: As with all phenacetin-containing products, avoid excessive or prolonged use.

Side Effects: Side effects are uncommon—nausea, constipation, and drowsiness have been reported.

A·H·ROBINS

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When the agitated businessman goes to work...

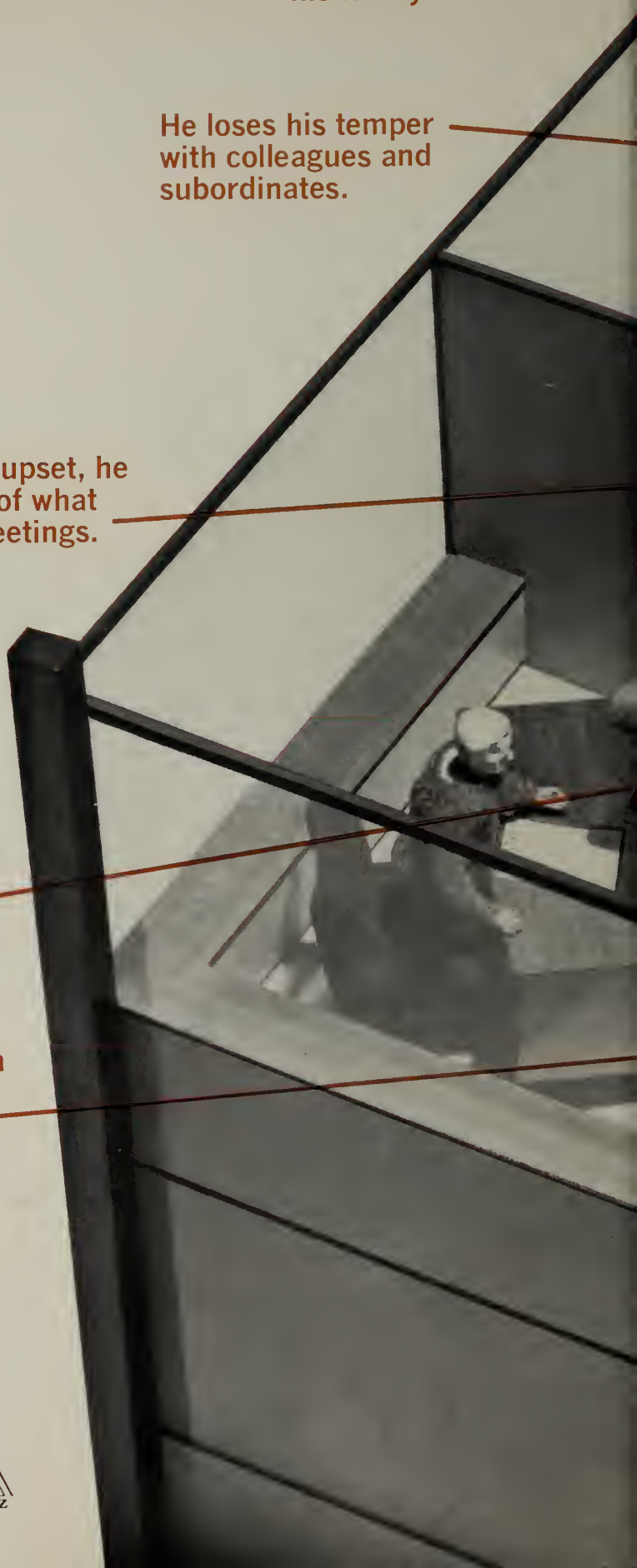
He goes home at night and takes it out on his family.

He loses his temper with colleagues and subordinates.

Emotionally upset, he misses half of what is said at meetings.

He is always fearful about his standing with the boss.

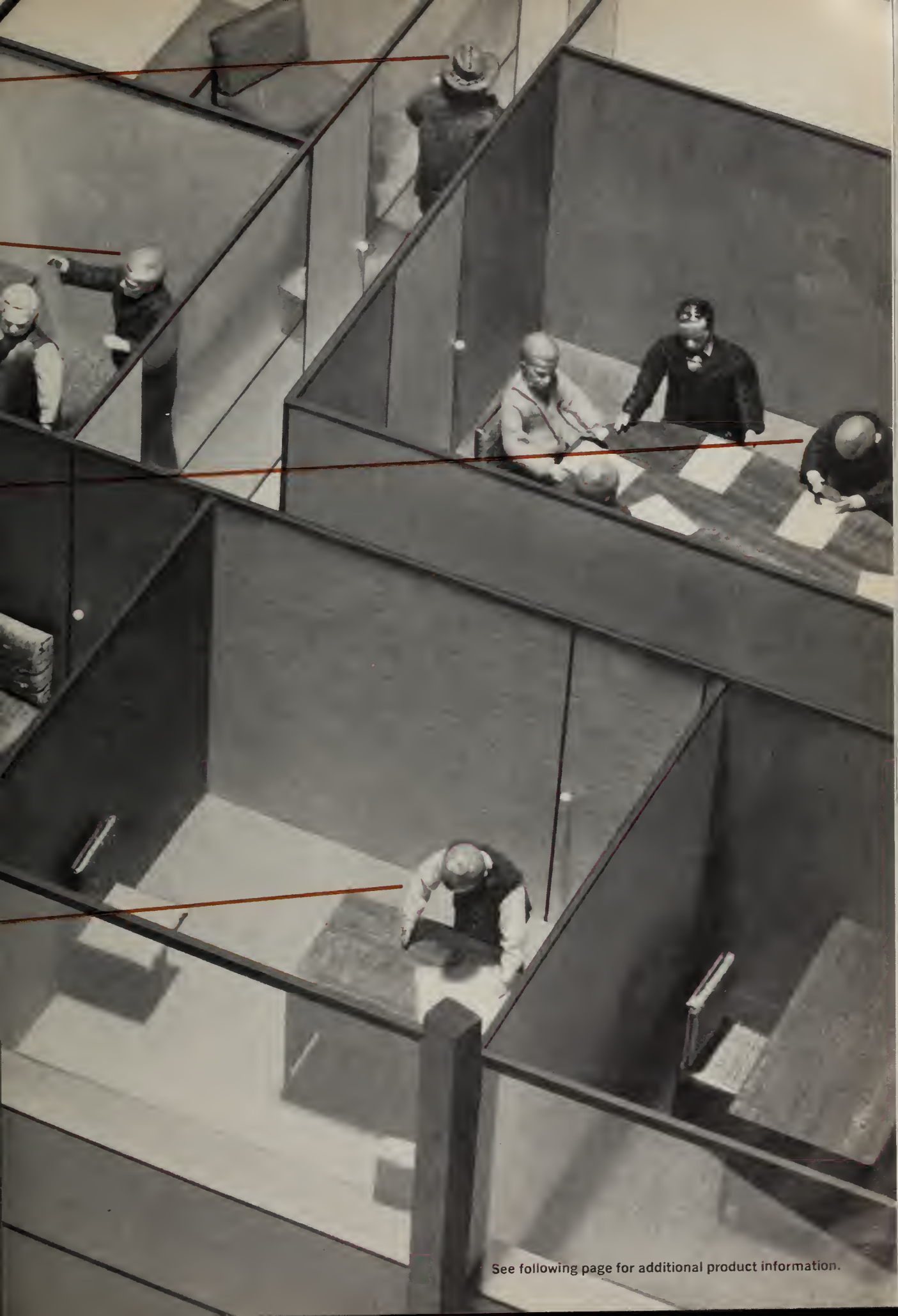
He just can't seem to settle down to his work.



for moderate to severe anxiety

Mellaril®
(thioridazine)
25 mg. t.i.d.

SANDOZ



See following page for additional product information.

When the agitated businessman goes to work...

Anxiety that seriously interferes with the individual's performance at work, at home, or in the community may be regarded as moderate to severe in degree.

Mellaril often recommends itself to the treatment of moderate to severe anxiety because it

- helps control the most frequent symptoms: marked tension, agitation, apprehension, restlessness, hypermotility
- often alleviates anxiety-induced somatic complaints
- frequently helps strengthen emotional resources
- helps the patient maintain realistic contact with environment, closer harmony with family

Thus, when you consider the anxiety moderate to severe... consider Mellaril.

Contraindications: Severely depressed or comatose states from any cause, and in association with or following MAO inhibitors; severe hypertensive or hypotensive heart disease.

Precautions: Hypersensitivity reactions (e.g., leukopenia, agranulocytosis) and convulsive seizures are infrequent. Pigmentary retinopathy has been observed where doses in excess of those recommended were used for long periods of time. May potentiate central nervous system depressants, atropine, and phosphorus insecticides. Where complete mental alertness is required, administer the drug cautiously and increase dosage gradually. In addition, orthostatic hypotension (especially in female patients) has been observed. Epinephrine should be avoided in treatment of drug-induced hypotension.

Side Effects: Pseudoparkinsonism and other extrapyramidal disorders are infrequent; drowsiness, especially in high doses early in treatment, may occur; nocturnal confusion, dryness of the mouth, nasal stuffiness, headache, peripheral edema, lactation, galactorrhea, and inhibition of ejaculation are noted on occasion; photosensitivity and other allergic skin reactions may occur but are extremely rare.

Before prescribing, see package insert for full product information.

for moderate to severe anxiety

Mellaril®
(thioridazine)
25 mg. t.i.d.

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mudrane®

for

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- ASTHMA
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The fast-disintegrating uncoated tablet gives relief in 15 minutes

Each tablet contains:

Potassium Iodide	195 mg.
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Ephedrine HCl	16 mg.

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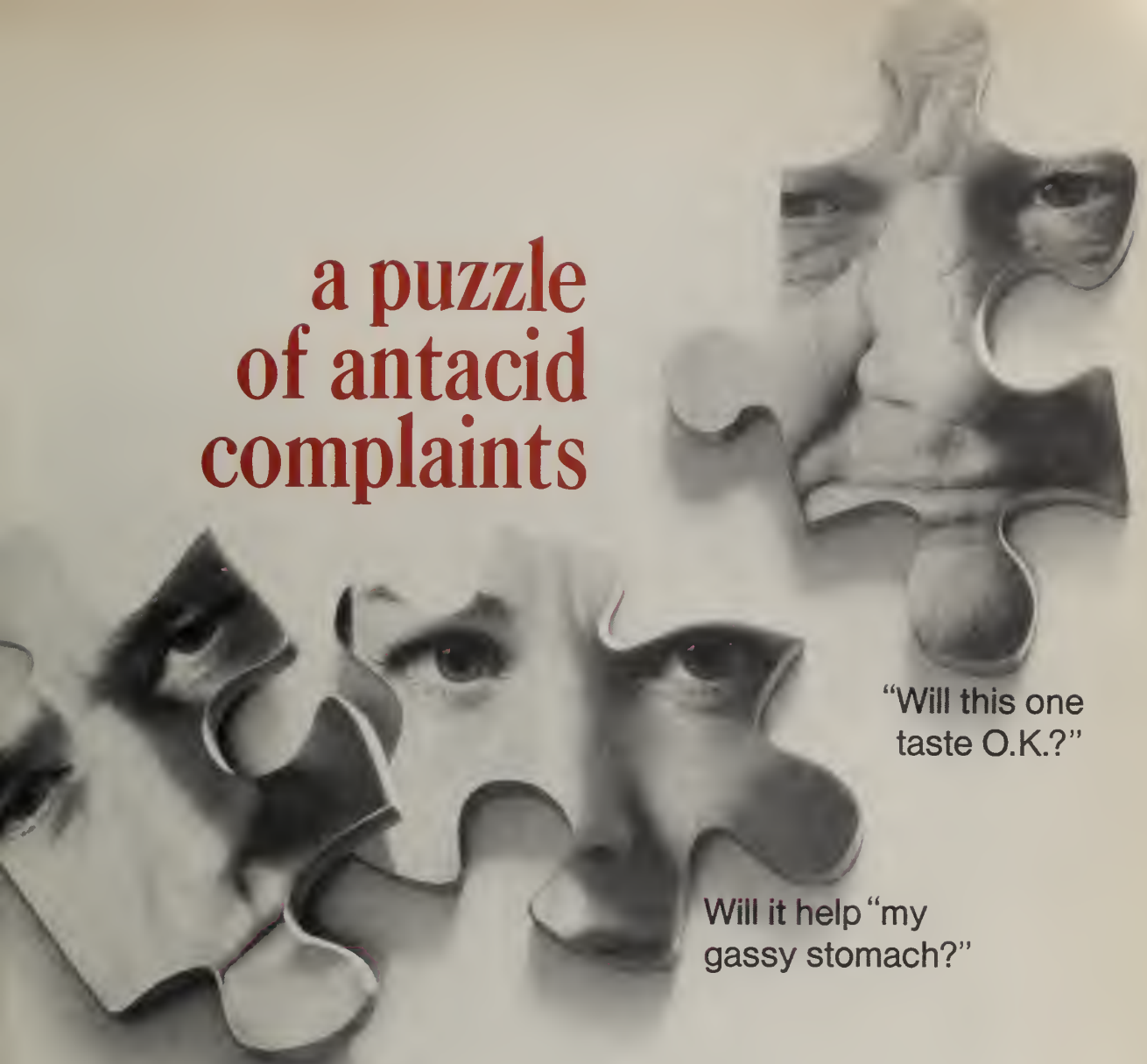
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References: 1. Hoon, J. R.: Arch. Surg. *93*:467 (Sept.) 1966.
2. Danhof, I. E., Personal communication.

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new evidence for TAO® (triacetyl-oleandomycin)

a macrolide antibiotic for the frequently seen respiratory infection in the office and for a problem pathogen* in the hospital.

*Staphylococcus aureus

study I[1]

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97.0%
of the organisms were susceptible to oleandomycin*

98.0%
of the patients responded favorably to TAO® (triacetyloleandomycin)

*In some cases more than one pathogenic organism was isolated from the patient.

study II[2]

Effect of oral therapy with TAO, erythromycin, and cloxacillin on the survival time of Rhesus monkeys after intravenous inoculations of lethal doses of staphylococci, phage type 80/81.
(8 monkeys in each group)

conclusion:

*Under the conditions of this study and the doses employed, it was found that

TAO® (triacetyl-oleandomycin)

was far superior to erythromycin, as was cloxacillin, a bactericidal agent, and of particular interest, "...bacteriostatic triacetyloleandomycin was as effective or perhaps superior to cloxacillin in preventing lethal (staphylococcal, phage type 80/81) infection."

*It should be pointed out that results obtained in an experimental study of this nature may not necessarily be directly extrapolated to the clinical situation as it pertains to man.

TAO Rx Information

INDICATIONS: Include streptococci, staphylococci, pneumococci and gonococci. Recommended for acute, severe infections where adequate sensitivity testing has demonstrated susceptibility to this antibiotic and resistance to less toxic agents. **CONTRAINDICATIONS AND PRECAUTIONS:** Not recommended for prophylaxis or in the treatment of infectious processes which may require more than ten days continuous therapy. In view of the possible hepatoxicity of this drug when therapy beyond ten days proves necessary, other less toxic agents should be used. If clinical judgement dictates continuation of therapy for longer periods, serial monitoring of liver profile is recommended, and the drug should be discontinued at the first evidence of any form of liver abnormality. When treating gonorrhea in which lesions of primary or secondary syphilis are suspected, proper diagnostic procedures, including darkfield examinations, should be followed. In other cases in which concomitant syphilis is suspected, monthly serological tests should be made for at least four months. Contraindicated in pre-existing liver disease or dysfunction, and in individuals hypersensitive to the drug. Although reactions of an allergic nature are infrequent and seldom severe, those of the anaphylactoid type have occurred on rare occasions. When used in streptococcal infections, therapy should be continued for ten days to prevent the development of rheumatic fever or glomerulonephritis. The use of antibiotics may occasionally permit overgrowth of nonsusceptible organisms. A resistant infection or superinfection requires re-evaluation of the patient's therapy. In the event such occurs with this drug the medication should be discontinued, and specific antibacterial and supportive therapy instituted.

References: 1. Isenberg, H. D.: Clinical Evaluation of Laboratory Guidance to Antibiotic Therapy; Health Laboratory Science (July) 1967. 2. Saslaw, S., Carlisle, H. N.: Studies on Therapy of Staphylococcal Infections in Monkeys. 1. Comparison of Cloxacillin, Triacetyloleandomycin and Erythromycin. Proc. Soc. Exp. Biol. & Med.: Vol. 125, No. 4 (Aug.-Sept.) 1967.

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Winthrop

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NASAL SPRAY

relieves
nasal symptoms
on contact

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Dextromethorphan hydrobromide	30 mg.
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Acetaminophen	325 mg.

Dosage: Adults—1 tablet, swallowed whole to preserve timed-release feature, in morning, midafternoon and at bedtime. Side effects: Occasional drowsiness, blurred vision, cardiac palpitations, flushing, dizziness, nervousness or gastrointestinal upsets. Precautions: The patient should be advised not to drive a car or operate dangerous machinery if drowsiness occurs. Use with caution in patients with hypertension, heart disease, diabetes or thyrotoxicosis.

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Galvanic skin resistance... one measure of the Valium (diazepam) effect in reducing the somatic response to acute stress[1,2]

The subjective value of Valium (diazepam) has been proved by many clinical and empirical evaluations. Now, also objective criteria such as used in other areas of scientific research have demonstrated the effectiveness of Valium (diazepam) in reducing certain somatic responses to acute stress.

Devised as a research tool in psychosomatic medicine, this interesting method records respiratory excursions, galvanic skin resistance, cardiac activity and finger pulse volume as physical responses to stress. These autonomic nervous system responses to a standardized stressor—a film dealing with life-threatening incidents—are continuously monitored on the polygraph.

This gives a graphic representation of changes which the body may undergo in reacting to stress. The application of modern statistical analysis to these graphs provides a yardstick by which the calming effect of Valium (diazepam) can be measured quantitatively.

Before prescribing, please consult complete product information, a summary of which follows:

Contraindications: Infants, patients with a history of convulsive disorders, glaucoma or known hypersensitivity to drug.

Warning: Not of value in the treatment of psychotic patients, and should not be employed in lieu of appropriate treatment.

Precautions: Limit dosage to smallest effective amount in elderly or debilitated patients (not more than 1 mg, one or two times daily initially) to preclude ataxia or oversedation, increasing gradually as needed or tolerated. As is true of all CNS-acting drugs, until correct maintenance dosage is established, advise patients against possibly hazardous procedures requiring complete mental alertness or physical coordination. Driving during therapy not recommended. In general, concurrent use with other psychotropic agents is not recommended. If such combination therapy is used, carefully consider individual pharmacologic effects—particularly with known compounds which may potentiate action of Valium (diazepam), such as phenothiazines, barbiturates, MAO inhibitors and other antidepressants. Advise patients against simultaneous ingestion of alcohol or other CNS depressants. Safe use in pregnancy not established. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Observe usual precautions in impaired renal or hepatic function. Periodic blood counts and liver function tests advisable in long-term use. Cease therapy gradually.

Side Effects: Side effects (usually dose-related) are fatigue, drowsiness and ataxia. Also reported: mild nausea, dizziness, blurred vision, diplopia, headache, incontinence, slurred speech, tremor and skin rash; paradoxical reactions (excitement, depression, stimulation, sleep disturbances, acute hyperexcited states, hallucinations); changes in EEG patterns during and after drug treatment. Abrupt cessation after prolonged overdosage may produce withdrawal symptoms (convulsions, tremor, abdominal and muscle cramps, vomiting, sweating) similar to those seen with barbiturates, meprobamate and chlordiazepoxide HCl.

Dosage—*Adults:* Mild to moderate psychoneurotic reactions, 2 to 5 mg b.i.d. or t.i.d.; severe psychoneurotic reactions, 5 to 10 mg t.i.d. or q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; muscle spasm with cerebral palsy or athetosis, 2 to 10 mg t.i.d. or q.i.d. *Geriatric patients:* 1 or 2 mg/day initially, increase gradually as needed and tolerated. (See *Precautions.*)

Supplied: Valium® (diazepam) Tablets, 2 mg, 5 mg and 10 mg; bottles of 50 and 500.

Roche Laboratories, Division of Hoffmann-La Roche Inc. Nutley, N.J. 07110

References: 1. Selesnick, S. T., and Clemens, T. L.: From research film "Motion Picture Films in Psychosomatic Research," available from Roche Laboratories. 2. Clemens, T. L., and Selesnick, S. T.: *Dis. Nerv. System,* *28*:98, 1967.

VALIUM® (diazepam) Roche®

useful for somatic symptoms of psychic tension



Indications: Tofrānil is recommended for the treatment of depressive states of diverse psychopathology.
Contraindications: The concomitant use of Tofrānil and monoamine oxidase inhibiting (M.A.O.I.) compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur. Potentiation of adverse effects can be serious or even fatal. An interval of at least 7 days after M.A.O.I. therapy has been discontinued should be allowed before Tofrānil may be substituted. Initial Tofrānil dosage should be low, increases should be gradual, and the patient's progress should be carefully observed.
Warning: Clinical reports have suggested that there may be a risk of teratogenesis associated with the use of this compound during the first trimester of pregnancy. Unless, in the opinion of the prescribing physician, the potential benefits outweigh the possible risks, Tofrānil should not be used during the first trimester of pregnancy.
Cardiovascular complications, including myocardial infarction and arrhythmias, have occasionally occurred in susceptible individuals. Patients with cardiovascular disease should be given the drug only under careful observation and in low dosage.
Precautions: Since suicide is always a possibility in severely depressed patients and one which may persist until significant remission occurs, such patients should be carefully supervised during early treatment with Tofrānil. Some severely depressed patients may also require hospitalization and/or concomitant electroconvulsive therapy.
Because of its anticholinergic effect, caution should be observed in prescribing Tofrānil for patients with increased intraocular pressure.
In rare instances, transient cardiac arrhythmias have occurred in hyperthyroid patients and in patients receiving thyroid medication when Tofrānil was added to the regimen.
Imipramine may block the pharmacologic activity of guanethidine and other related adrenergic neuron-blocking agents.
The drug is not recommended at the present time in patients under 12 years of age.
Adverse Reactions: Dryness of the mouth, tachycardia, constipation, disturbances of accommodation, sweating, dizziness, weight gain, urinary frequency or retention, nausea and vomiting, peripheral neuritis, mild parkinson-like syndrome, tremors, rare cases of falling in elderly patients, confusional states (with such symptoms as hallucinations and disorientation), activation of psychosis in schizophrenics and agitation (includ-

When a milestone in life is marred by depression...



Often in the mind of the lonely, widowed, depression-prone individual, she's not gaining a daughter...she's losing a son. The occasion may be marred by such symptoms of depression as feelings of sadness, incapacity, helplessness and hopelessness.

In about 3 out of 4 cases, Tofrānil relieves symptoms of primary depression.

As maintenance therapy in primary depressive illness, it helps prevent relapse.

Although toxic reactions severe enough to require discontinuation of Tofrānil are uncommon, in patients with cardiovascular disease, thyroid disorders, increased intraocular pressure, or in those receiving anticholinergics (including antiparkinsonism agents), the special precautions listed in the Prescribing Information should be carefully observed. The use of Tofrānil in patients receiving M.A.O.I.'s is contraindicated.

Tofrānil® imipramine hydrochloride
Geigy

ing hypomanic and manic episodes) which may require dosage reduction and/or addition of a tranquilizer or temporary discontinuation of the drug, epileptiform seizures, orthostatic hypotension and substantial blood pressure fall in hypertensive patients, purpura, transient jaundice, bone marrow depression including agranulocytosis, sensitization and skin rash including photosensitization, eosinophilia, and mild withdrawal symptoms on sudden discontinuation after prolonged treatment with high doses. Occasional hormonal effects (impotence, decreased libido, and estrogenic effects) may be observed. Atropine-like effects may be more pronounced (e.g. paralytic ileus) in susceptible patients and in those using anticholinergic agents (including antiparkinsonism drugs).
Outpatient Adult Dosage: Initially, 75 mg. daily, increased, if necessary, to 150 or 200 mg. Maintenance dosage may be lower, 50 to 150 mg. daily, if possible.
Geriatric and Adolescent Dosage: Initially, 30 or 40 mg. daily, which may be increased according to response and tolerance. It is usually unnecessary to exceed 100 mg. daily.
A lag in therapeutic response, lasting from a few days to a few weeks, should be expected. When dosage recommendations are already being followed, increasing the dosage does not normally shorten this latency period and may increase the incidence of adverse reactions.
Availability: Tofrānil: Round tablets of 25 and 50 mg.; triangular tablets of 10 mg. for geriatric and adolescent use; and ampuls, each containing 25 mg. in 2 cc. for I.M. administration.
(B)46-850-C
For complete details, please refer to the full Prescribing Information.

Geigy Pharmaceuticals
Division of Geigy Chemical Corporation
Ardsley, New York 10502



in alcoholism:



B and C vitamins aid therapy. Therapeutic amounts of B and C vitamins can be important in the management of the alcoholic patient. In alcoholism, as in many chronic illnesses, STRESSCAPS vitamins aid therapy.

Stresscaps®

Stress Formula Vitamins Lederle

FOR PROFESSIONAL RECOMMENDATION ONLY

Each capsule contains:	
Vitamin B_1 (as Thiamine Mononitrate)	10 mg
Vitamin B_2 (Riboflavin)	10 mg
Vitamin B_6 (Pyridoxine HCl)	2 mg
Vitamin B_{12} Crystalline	4 mcgm
Vitamin C (Ascorbic Acid)	300 mg
Niacinamide	100 mg
Calcium Pantothenate	20 mg

Recommended intake: Adults, 1 capsule daily, for the treatment of vitamin deficiencies. Supplied in decorative "reminder" jars of 30 and 100; bottles of 500.

LEDERLE LABORATORIES, A Division of American Cyanamid Company, Pearl River, New York

691—6—3942

Lederle

If hypothyroidism leaves your patient feeling like this...

consider

LETTER®

(SODIUM LEVOTHYROXINE, ARMOUR) TABLETS

Synthetic Thyroid Replacement Therapy



Letter® provides all the advantages of the synthetically pure chemical sodium levothyroxine. Dosage is precise and potency is consistently uniform.

and

Letter® is micronized to provide maximum opportunity for full absorption and clinical response.

In addition Letter® is distinctively color coded with an identifying number stamped on each tablet to provide accurate dosage control.

Indications: Hypothyroid conditions. **Contraindications:** Thyrotoxicosis, acute myocardial infarctions unless associated with hypothyroidism. In hypothyroidism with hypoadrenalism coadministration of corticoids with LETTER® is recommended. **Precautions and Side Effects:** Excessive dosage may result in diarrhea, cramps, palpitation, nervousness, rapid pulse, sweating. If symptoms appear, discontinue medication for several days, then reinstitute at a lower level. Since myxedema patients with heart disease may suffer seriously from abrupt increases in dosage, caution should be exercised in adjusting dosage. **Dosage:** Generally, the initial adult dosage is 0.1 mg. daily. This may be increased in small increments every one to three weeks until proper metabolic balance is achieved. **Available:** Bottles of 100 tablets, in six potencies: 0.025 mg. (violet), 0.05 mg. (peach), 0.1 mg. (pink), 0.2 mg. (green), 0.3 mg. (yellow), and 0.5 mg. (white).

ARMOUR PHARMACEUTICAL COMPANY • CHICAGO, ILLINOIS



treat one . . . six people benefit

The brunt of senility falls on the family as much as the patient. But usually within one or two days, 'Thorazine' can control senile anxiety and fear . . . dispel the confusion shown by nighttime wandering and chattering . . . restore appetite and interest in personal grooming. Treat the senile patient with 'Thorazine'—the whole family can benefit.

Before prescribing, see complete information, including adverse effects reported with phenothiazines and symptoms and treatment of overdosage, in SK&F literature or *PDR*. The following is a brief precautionary statement.

Contraindications: Comatose states or the presence of large amounts of C.N.S. depressants.

Precautions: Potentiation of C.N.S. depressants may occur (reduce dosage of such agents when used concomitantly). Use with caution in patients with chronic respiratory disorders. Antiemetic effect may mask overdosage of toxic drugs or obscure other conditions. Administer in pregnancy only when necessary. Because of possible drowsiness use cautiously and warn patients who operate vehicles or machinery.

Adverse Reactions: Drowsiness; dry mouth; nasal congestion; constipation; amenorrhea; miosis; mild fever; weight gain; hypotensive effects, sometimes severe with I.M. administration; epinephrine effects may be reversed; dermatological reactions; parkinsonism-like symptoms on high dosages (in rare instances, may persist); lactation and moderate breast engorgement (in females on high dosages); and less frequently, cholestatic jaundice (use cautiously in patients with liver disease). Adverse reactions occurring rarely, include: mydriasis; agranulocytosis; skin pigmentation; epithelial keratopathy; lenticular and corneal deposits (after prolonged substantial doses).

Available: Tablets, 10 mg., 25 mg., 50 mg., 100 mg. and 200 mg.; Spansule® capsules, 30 mg., 75 mg., 150 mg., 200 mg. and 300 mg.; Injection, 25 mg./cc.; Syrup, 10 mg./5 cc.; Suppositories, 25 mg. and 100 mg.

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Smith Kline & French Laboratories

SK&F

in senile agitation . . . Thorazine® brand of chlorpromazine

Sources of sinus headache



REGION OF SENSATION

FRONTAL SINUS—headache localized over the frontal region, commonly beginning in the morning. Eyes painful to touch, may be slight swelling over eyes, puffy eyelids.
SPHENOID SINUS—most difficult to diagnose. Headache or sense of pressure over vertex. May be pain in ear or mastoid region.



MAXILLARY SINUS—headache and tenderness under eyes at top of cheekbones, usually starting in early afternoon. Possible swelling of cheek. Pain in jaw and back teeth.
ETHMOID SINUS—headache, tightness in upper part of bridge of nose and well down its sides. Pain, deep-seated behind the eyes and in inner corners of the eyes.



TURBINATES—pain along medial and lateral walls of the inside of the nose. Headache in frontal region, along supraorbital ridge, a feeling of fullness in the head.

Source of symptomatic relief

SINUTAB



Sinus headache is often a reflection of congestion in the nasal mucosa. The pain that results in the various regions of the head may help in determining the particular structure(s) responsible. The Sinutab formula is specifically designed for symptomatic relief of sinus headache and nasal congestion. **Sinutab** *not only* **provides an effective decongestant** — to reduce mucosal engorgement and facilitate freer breathing — *and* **an antihistamine** — to help reduce edema and excessive secretions — but it *also* provides, in contrast to other leading decongestants, **two analgesics** for immediate relief of pain and discomfort.

And, too, the Sinutab dosage schedule of 2 tablets, *q.* 4 *h.* permits controlled and continuing analgesic relief.

Adverse Reactions: Epigastric distress, drowsiness, dizziness, insomnia and nervousness.

Precautions: Instruct patients not to drive or operate machinery if drowsiness occurs. Use with caution in patients with thyroid disease, heart disease, hypertension, diabetes or kidney disease. Excessive dosage or prolonged use may cause kidney damage.

Dosage: Adults—2 tablets every 4 hours.

Each tablet contains 150 mg. acetaminophen, 150 mg. phenacetin, 25 mg. phenylpropanolamine HCl, and 22 mg. phenyltoloxamine citrate.

SPECIFIC FORMULA **SINUTAB®** FOR SINUS HEADACHE

Also Available on Prescription:

Sinutab® with Codeine.* Each tablet contains 15 mg. (¼ gr.) codeine phosphate (Warning: May be habit forming), 150 mg. acetaminophen, 150 mg. phenacetin, 25 mg. phenylpropanolamine HCl, and 22 mg. phenyltoloxamine citrate.

Sinutab® Pediatric Suspension. Each 5 ml. teaspoonful contains 300 mg. acetaminophen, 12.5 mg. phenylpropanolamine HCl, and 10 mg. phenyltoloxamine citrate.

*Subject to Federal Narcotics Regulations.

For 1 complimentary bottle of 30 Sinutab tablets, write: J. S. Travis, Warner-Chilcott Laboratories, Morris Plains, N.J. 07950. Please mention this journal.

S-IN-81-4C

WC **WARNER-CHILCOTT** Morris Plains, N.J.



"Yes, Doctor, the pain is gone."

'EMPIRIN'® COMPOUND with CODEINE PHOSPHATE gr. 1/2 No. 3

Each tablet contains: Codeine Phosphate gr. ½ (Warning – May be habit forming), Phenacetin gr. 2½, Aspirin gr. 3½, Caffeine gr. ½.

■ Despite introduction of synthetic substitutes, efficacy of 'Empirin' Compound with Codeine remains unchallenged.

BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, N.Y.



Indications: Hypertension and many types of edema involving retention of salt and water.
Contraindications: Hypersensitivity and most cases of severe renal or hepatic disease.
Warning: With the administration of enteric-coated potassium supplements, which should be used only when adequate dietary supplementation is not practical, the possibility of small bowel lesions (obstruction, hemorrhage, and perforation) should be kept in mind. Surgery for these lesions has frequently been required and deaths have occurred. Discontinue enteric-coated potassium supplements immediately if abdominal pain, distention, nausea, vomiting, or gastrointestinal bleeding occur. Use with caution in pregnant patients, since the drug may cross the placental barrier and adverse reactions which may occur in the adult (thrombocytopenia, hyperbilirubinemia, altered carbohydrate metabolism, etc.) are potential problems in the newborn.
Precautions: Antihypertensive therapy with Hygroton should always be initiated cautiously in postsympathectomy patients and in patients receiving ganglionic blocking agents or other potent antihypertensive drugs, or curare. Reduce dosage of concomitant antihypertensive agents by at least one-half. Barbiturates, narcotics or alcohol may potentiate hypotension. Because of the possibility of progression of renal damage, periodic determination of the BUN is indicated. Discontinue if the BUN rises or liver dysfunction is aggravated. Hepatic coma may be precipitated. Electrolyte imbalance, sodium and/or potassium depletion may occur. If potassium depletion should occur during therapy, Hygroton should be discontinued and potassium supplements given, provided the



Did Dorothy Larson show you her ankles in private?

Now she shows them in public.

Hygroton® Geigy
chlorthalidone

Your office examination confirmed Mrs. Larson's ankle edema.

You prescribed Hygroton—to get rid of the edema.

And you found that Hygroton is not only effective; it frequently costs less than other equivalent therapy.

A nice way to treat the Mrs. Larsons in your practice.

Hygroton therapy may mean troublesome side effects for some patients. A summary of the prescribing information is shown below.

ient does not have marked oli-
ia.
e special care in cirrhosis or
ere ischemic heart disease and
atients receiving corticoste-
ds, ACTH, or digitalis. Salt re-
ction is not recommended.
erse Reactions: Nausea, gastric
ation, vomiting, anorexia, con-
ation and cramping, dizziness,
akness, restlessness, hyperglyc-
nia, hyperuricemia, headache,
scle cramps, orthostatic hypo-
tension, aplastic anemia, leuko-
penia, thrombocytopenia, agranu-
locytosis, impotence, dysuria,
transient myopia, skin rashes, urti-
caria, purpura, necrotizing angiitis,
acute gout, and pancreatitis when
epigastric pain or unexplained G.I.
symptoms develop after prolonged
administration. Other reactions re-
ported with this class of compounds
include: jaundice, xanthopsia,
paresthesia, and photosensitization.
Average Dosage: 50 or 100 mg. with
breakfast daily or 100 mg. every
other day.
Availability: White, single-scored
tablets of 100 mg. and aqua tablets
of 50 mg., in bottles of 100 and 1000.
(B)R46-230-D
**For full details, please see the com-
plete prescribing information.**

Geigy Pharmaceuticals
Division of
Geigy Chemical Corporation
Ardsley, New York 10502

HY-5576

100 mg. Hygroton®
chlorthalidone

and new 50 mg. Hygroton



For the Dorothy Larsons in your practice, you can prescribe Hygroton either way.

Hygroton 50 mg. offers convenience for your patients who are halving the 100 mg. tablets or taking one every other day.

Geigy

Please see preceding pages for prescribing summary.

Geigy Pharmaceuticals
Division of Geigy Chemical Corporation
Ardsley, New York 10502



Photo professionally posed.

No injection after all!

This penicillin produces high, fast levels—orally.

Pen•Vee® K is usually so rapidly and completely absorbed that therapeutic penicillin levels are attained within 15 to 30 minutes. Thus it can often obviate the need for penicillin injections. The higher serum levels produced generally last longer than with those of oral penicillin G.

Indications: Infections susceptible to oral penicillin G: propnylaxis and treatment of streptococcal infections; treatment of pneumococcal, gonococcal, and susceptible staphylococcal infections; prophylaxis of rheumatic fever in patients with a previous history of the disease.
Contraindications: Infections caused by nonsusceptible organisms; history of penicillin sensitivity.
Warnings: Acute anaphylaxis (may prove fatal unless promptly controlled) is rare but more frequent in patients with previous penicillin sensitivity, bronchial asthma or other allergies. Resuscitative (epinephrine, aminophylline, pressor amines) and supportive (antihistamines, methylprednisolone sodium succinate) drugs should be readily available. Other rare hypersensitivity reactions include nephropathy, hemolytic anemia, leucopenia and thrombocytopenia. In suspected hypersensitivity, evaluation of renal and hematopoietic systems is recommended.
Precautions: In suspected staphylococcal infections, perform proper laboratory studies including sensitivity tests. If overgrowth of nonsusceptible organisms occurs (constant observation is essential), discontinue penicillin and take appropriate measures. Whenever allergic reactions occur, withdraw penicillin unless condition being treated is considered life threatening and amenable only to penicillin. Penicillin may delay or prevent appearance of primary syphilitic lesions. Gonorrhea patients suspected of concurrent syphilis should be tested serologically for at least 3 months. When lesions of primary syphilis are suspected, dark-field examination should precede use of penicillin. Treat beta-hemolytic streptococcal infections with full therapeutic dosage for at least 10 days to prevent rheumatic fever or glomerulonephritis. In staphylococcal infections, perform surgery as indicated.
Adverse Reactions: (Penicillin has significant index of sensitization): Skin rashes, ranging from maculopapular eruptions to exfoliative dermatitis; urticaria; serum sickness-like reactions, including chills, fever, edema, arthralgia and prostration. Severe and often fatal anaphylaxis has been reported (see "Warnings").
Composition: Tablets—125 mg. (200,000 units), 250 mg. (400,000 units), 500 mg. (800,000 units); Liquid—125 mg. (200,000 units) and 250 mg. (400,000 units) per 5 cc.

Wyeth Laboratories Philadelphia, Pa.

ORAL PEN•VEE® K

(potassium phenoxymethyl penicillin) Wyeth®



Now...twice as much as before in each teaspoon

400,000 units of potassium penicillin V per teaspoonful

New...V-Cillin K®, Pediatric, 250 mg.

Potassium Phenoxymethyl Penicillin

Lilly

Additional information available to physicians upon request. Eli Lilly and Company, Indianapolis, Indiana 46206.

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The Virginia
MEDICAL MONTHLY

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Guest Editorial . . . .

A Hospital Staff Nurse Speaks

THE SURGEON GENERAL of the United States in an article titled "The Surgeon General looks at Nursing" (American Journal of Nursing, January, 1967, stated: "Nursing needs outside help." It is because I agree with this statement that I venture to put my feelings in writing. In this article it is necessary to say that all statements are the result of my own observations and experiences as a member of a hospital's nursing staff for the past twenty years.

During these aforementioned years I have tried to isolate the basic problem in nursing, because the problems in nursing and their inevitable effects on society are now of national concern. I say basic problem since usually when there are many problems present, there is one that when solved tends to solve other related problems.

What then are some of the undeniable characteristics of this elusive basic problem?

A. It cannot be solved by money, nor working hours, nor education alone, or in many cases it would be solved.

B. It cannot be solved by an increase in the number of nurses alone, for many recent surveys point up the fact that an adequate supply of nurses does not always insure better patient care.

C. It affects the hospital's nursing staff more than it affects other groups of nurses such as office nurses, school nurses, nursing educators, etc.

What then could be our basic problem that if solved could in turn solve the other problems mentioned? Could it be "lack of recognition"? Do we not hear this complaint expressed every day by hospital nurses and in retrospect, have we not heard it expressed continually since Florence Nightingale's time—in fact since the very inception of nursing as a profession? Is it not a problem we are unable to solve alone?

To those of you who would argue that we have received appreciable recognition let me hasten to ask, "Have the members of the hospital nursing staff received this recognition or could you be speaking of the nursing educators? There is an important difference here. Nursing educators have been accused of seeking status and whether they have sought it or not, I feel they have been fairly successful in obtaining it, but it has been such a singular success that I believe it may possibly have had some overall undesirable effects on nursing.

As an example, I feel that nursing educators are doing a commendable job of educating the ideal nurse but the flaw is that there is no ideal hospital environment in which this ideal nurse may practice. Yes, it has been a singular success! The fact that no ideal hospital environment exists is proof that the hospital staff nurse is still lacking in recognition.

For further proof let me quote from the reference manual of the Joint Commission on Hospital Accreditation.

> " . . . . the Joint Commission represents five medical and health organizations in the United States and Canada and has a board composed of 20 commissioners. These organizations, and the number of commissioners representing each, are:
>
> American College of Surgeons 3
> American College of Physicians 3
> American Hospital Association 7
> American Medical Association 6
> Canadian Medical Association 1
>
> "Its main purpose . . . . is to improve the quality of patient care in hospitals. Its method of fulfilling this purpose is to establish minimum standards of quality for patient care and then invite all hospitals and physicians offering care to the sick to meet or surpass those standards . . . . The following outlines the basic principles necessary for accreditation of a hospital by the Joint Commission. They are arranged in three sections according to the major divisions of a hospital.
>
> I. Administration—et cetera
>
> II. Medical Staff—et cetera
>
> III. Nursing"

Nursing alone has no representation among the members listed.

Would recognition solve our basic problem? I think the chain reaction, while it might be slow, would be startling! This would be representation of a large majority of nurses since approximately 70 percent of all nurses are hospital staff nurses. Problems would be presented and solved more realistically. Is it not true that today there is more understanding of nurses problems between the hospital staff nurses, physicians

and administrators than there is between the hospital staff nurses and nursing educators?

Another aspect which must be considered is that hospital nurses constitute a majority, and need only to become organized to make demands that will be met. Would it not be more sensible to offer them the representation that would be more beneficial to all concerned than to wait until these positions are achieved by demand? Otherwise would there not be more wounds to heal?

If you would ask me to what positions I refer I would list the following:

1. Appoint the Director of Nurses to the hospital Board of Directors. This recommendation was also made in a fairly recent Life magazine editorial.

2. Invite representation on the Joint Commission of Accreditation from Nursing Service Administration through the American Nurses Association.

3. Have a Nurse Consultant on all hospital construction. She should be recommended under the Hill Burton Act and if paid as other consultants (as she should be), she could afford to become increasingly more knowledgeable and valuable. I see here a much needed specialization by an adept few.

4. Representation on all national, state and local committees that have to do with health care should include a fair number of qualified hospital staff nurses who have been actively employed in a hospital for a minimum of five consecutive years.

Be assured that the Nursing Service has qualified members for representation. Many of our Directors of Nursing Service, assistants and staff members are as well educated as any in the nursing profession.

Yes, nursing needs outside help, and it needs it now if the dedication of the nursing profession is to be preserved and if there is to be a united team in charge of the nation's health.

LILLIAN T. STRAWN, R.N.

1521 West Laburnum Ave.
Richmond, Va.

Esophageal Atresia

EDWIN L. WILLIAMS, M.D.
ARNOLD M. SALZBERG, M.D.
Richmond, Virginia

The treatment of congenital esophageal atresia is difficult and usually complicated. Nevertheless, the mortality in recent years, in this series of 32 cases, has been reduced to 45%.

THE VARIOUS TYPES of esophageal atresia comprise an important segment of those congenital abnormalities that produce respiratory distress in the newborn. Surgical treatment is complicated by prematurity, other crippling anomalies and pneumonia. Nevertheless, this therapeutic challenge has been met by thoracic and pediatric surgical pioneers and forms an exciting chapter in neonatal management.

Clinical Material

Thirty-two newborns with esophageal atresia, with and without tracheoesophageal fistula, were seen at the Medical College of Virginia Hospitals during the 13 year period between January 1, 1954, and January 1, 1967. (Table 1)

Table 1
Esophageal Atresia (MCVH 1954-1966)

No. of cases	32
Type of atresia	
Prox. atresia-distal te fist.	31 (97%)
Prox. atresia-no te fist.	1 (3%)

From the Pediatric Surgical Service and the Division of Thoracic Surgery, Department of Surgery, Medical College of Virginia, Richmond. Presented at the Virginia Surgical Society Meeting, Staunton, April 29, 1967.

Anatomically, 31 babies had proximal atresia with distal fistula; one patient had atresia without fistula.

Historically, pregnancy was complicated in six mothers by polyhydramnios. (Table 2)

Table 2
Complications During Gestation (7 Patients)

Polyhydramnios	6
Premature delivery (1 month)	4
Preeclampsia	2
Ruptured membranes	1
Pyelonephritis	1

A third of the infants had severe associated anomalies and eight of these were premature. Unfortunately, the combination of multiple anomalies and prematurity is frequent. The cardiovascular and genitourinary systems were most often involved and multiple system abnormalities were not rare. (Table 3)

Table 3
Associated Severe Anomalies in 11 Patients

1. Cardiovascular: IVSD 2; IASD 1; Patent Ductus 1; Hypoplasia Right Atrium 1; Pulmonary Atresia 1; Transposition Aorta 2; Congenital Cyanotic Heart Disease 1.
2. Genitourinary: Hydroureter and Hydronephrosis 2; Absent Kidneys 2; Multicystic Kidney 1.
3. Gastrointestinal: Imperforate Anus 3; Duodenal Atresia 1.
4. Respiratory: Tracheomalacia 2; Cleft Palate 1; Choanal Atresia 1; Hypoplasia Mandible 1; Pierre-Robin Syndrome 1.
5. Central Nervous System: Mongolism 1.

The diagnosis of esophageal atresia was made from birth to six days of age on the basis of the various symptoms and signs of the usual clinical picture which included dyspnea, tachypnea, cyanosis, profuse bubbling and sometimes bile stained oropharyngeal secretions, regurgitation of feedings, respiratory distress, a distended abdo-

men, and finally radiographic confirmation. (Table 4)

TABLE 4
ESOPHAGEAL ATRESIA WITH TRACHEO-ESOPHAGEAL FISTULA

SYMPTOMS AND SIGNS

1. Profuse bubbling respirations (with or without bile)
2. Coughing, choking, cyanosis—respiratory distress
3. Regurgitation of feedings
4. Distended abdomen

DIAGNOSIS

1. Thoraco-abdominal x-rays
2. Passage of 8-10 F cath into pouch
3. Dye studies (½ cc)

A Technique for Division of the Tracheoesophageal Fistula and Esophagoesophagostomy

In the operating room, the newborn is taken from the isolette and placed on a heated mattress. An endotracheal tube is inserted under local anesthesia and general anesthesia follows. The electrocardiographic electrodes are applied and the rectal thermometer inserted. The gastrostomy tube is connected to straight drainage.

The infant is placed in the left lateral decubitus position with the right side up and tilted forward. The child is then strapped in this position. At this time it will be noted that a cut-down will run best in the right upper extremity since the other extremities are somewhat compressed by the child's position.

An incision is made commencing at the lateral border of the sacrospinalis muscle and proceeds beneath the tip of the scapula and ends 1 cm. below and lateral to the right nipple. The incision is carried through the muscles of the chest wall, the scapula elevated and ribs counted. The fourth rib is then carefully removed in a subperiosteal manner from its angle to the costochondral junction.

An incision is made in the periosteum of the rib bed and deepened down through the endothoracic fascia to the glistening parietal pleura. Traction hemostats are placed on the periosteum and endothoracic fascia and the extrapleural plane developed by blunt dissection. Utmost care must be exercised to avoid transecting the pleura at the apex of the chest, the angle of the ribs, and the azygos vein. The azygos vein is doubly ligated and divided and the mediastinal segment is taken along with the parietal pleura.

The extrapleural plane must be developed to the mediastinum anteriorly and posteriorly and to the diaphragm inferiorly; this exposure will mimic that obtained by the transpleural approach.

The mediastinal level of the azygos vein and the vagus nerve lead to the tracheoesophageal fistula. The proximal end of the distal esophagus and its junction with the trachea is carefully dissected and visualized clearly. Traction on the distal esophagus is maintained with a pediatric right angle clamp or an encircling 2-0 silk ligature. Care must be taken to avoid downward stripping of the esophageal blood supply by this maneuver.

An atraumatic 5-0 arterial silk suture is placed through the lower edge of the junction of the tracheoesophageal fistula on its esophageal side and the fistula divided in stages, suturing as the transection proceeds. After the fistula is partially divided, a 5-0 traction suture is placed through one lateral angle of the distal esophagus and with complete division the other lateral angle of the esophagus is similarly transfixed with a through and through 5-0 silk suture. The running 5-0 silk suture on the tracheal end of the tracheoesophageal fistula is continued back to its origin and tied.

The distal esophagus is gently placed on traction through the two silk sutures and a #8 catheter is inserted into the distal esophagus and threaded into the stomach to dilate this segment and rule out distal obstruction.

Dissection is then directed toward the proximal esophagus which must be mobilized into the lower neck. If difficulty is encountered in recognizing this, the upper pouch catheter is gently moved by the anesthesiologist and the structure will be recog-

nized promptly. A transfixion suture is placed through the tip of the thick proximal pouch for traction. Dissection of the proximal pouch is continued around the entire circumference except at its rather firm attachment to the trachea. This attachment is firmer at the tip of the pouch than it is more proximally. This area is the last to be dissected and should be divided very carefully under good exposure with good traction on the proximal esophagus so that the plane between the two structures is completely visualized. If any doubt exists, the dissection must be kept on the esophageal wall to avoid penetration of the trachea.

The tip of the pouch is then removed; this tip will contain the traction suture. Suction is applied to the saliva contained therein from above and below and this is cultured.

The two sutures which were used as angle traction sutures for the distal esophagus are then employed to approximate the angles of the upper pouch to the lower pouch. Interrupted sutures are used for the posterior wall anastomosis and this is usually performed by bisecting the posterior half with one suture and then placing one or two sutures between the middle suture and the two angle sutures on either side.

The #8 suction catheter which had been in the proximal pouch is threaded into the distal esophagus and the anterior wall of the anastomosis is handled in a similar manner with interrupted sutures of 5-0 arterial silk so that the entire circumference is approximated by 8 to 10 sutures. The catheter is removed.

A suture is taken in the proximal esophagus and hooked into the paravertebral fascia at a lower level in order to remove tension on the suture line.

A #12 or #14 intercostal tube is inserted extrapleurally, connected to underwater seal and the chest wall closed.

Approximately 50 to 75 c.c. of blood is used during the operative procedure. (Figure 1)

Results

There were 12 survivors in 32 patients during this 13 year interval—a mortality of 65%. The mortality in the first seven year period, from 1954 through 1960, with 15 newborns, was 80% while during the

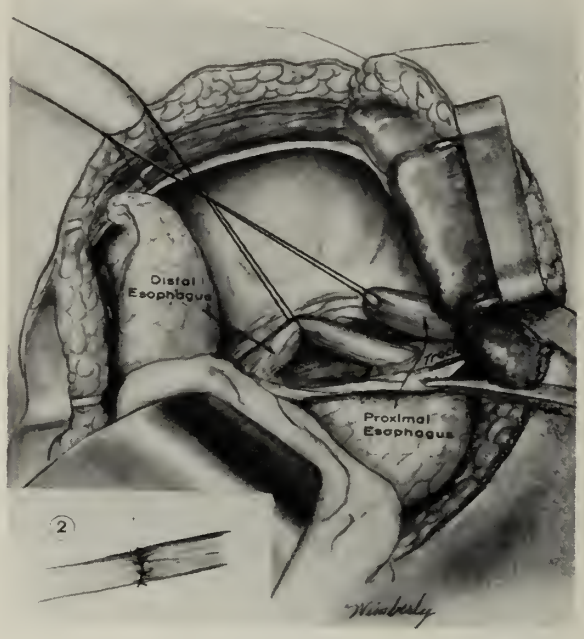


Fig. 1. Operative view through the transpleural approach. Insert shows the completed one-layer anastomosis.

past six years, from 1961 through 1966, with 17 infants, the mortality has been lowered to 45%. (Table 5)

Table 5

Esophageal Atresia
M.C.V. 1954-1966

	1954-1966 (13)	1954-1960 (7)	1961-1966 (6)
Alive	12 (35%)	3 (20%)	9 (55%)
Dead	20 (65%)	12 (80%)	8 (45%)
Total	32	15	17

Morbidity and mortality have been analyzed according to the presence of anomalies, the weight of the patient and the operative technique. The mortality was 80% in eleven newborns with crippling major anomalies. (Table 6) There was an

Table 6

Survival Related to Presence of Anomalies

Total Cases	Cases with Anomalies	Living	Dead
32	11 (33%)	2 (20%)	9 (80%)

70% of anomalies occurred in prematures
50% of prematures had anomalies

85% mortality in neonates four pounds and less while in newborns weighing six pounds and more it was a more reasonable 40%. (Table 7)

Table 7

Survival Related to Weight of Child

	4 lbs.	4-5 lbs.	5-6 lbs.	6 lbs.
Living	1(15%)	3(40%)	3(30%)	5(60%)
Dead	6(85%)	4(60%)	7(70%)	3(40%)

sis was reviewed in relation to survival and complications. The majority of the survivors had a one layer retropleural esophagoesophagostomy. (Table 9)

Anastomotic leaks occurred in three cases from the fourth to the sixth postoperative day and all occurred with primary transpleural two-layered repairs, (Haight) and in each instance the cases terminated fatally

Table 8

Survival Related to Weight and Operative Approach

Weight	Primary Retropleural		Primary Transpleural		Staged		Non-Op	
	Living	Dead	Living	Dead	Living	Dead	Living	Dead
4 lbs. (6)	0	0	0	2	1	1	0	1
4-5 lbs. (8)	2	3	0	0	1	2	0	0
5-6 lbs. (10)	2	1	0	4	1	1	0	1
6 lbs. (8)	3	2	2	0	0	1	0	0
Total 32	7	7	2	6	3	5	0	2

In relating survival to weight and operative approach, the mortality was 100% in patients weighing four pounds or less treated by primary anastomosis. In the heavier infants, the primary retropleural approach would seem to give better results than the transpleural approach. A reasonable survival has followed operative staging in those neonates who are badly premature or afflicted with other serious anomalies. (Table 8)

The technique of exposure and anastomo-

from intrapleural empyema. There were four strictures of whom three are presently alive. All were treated by dilatation over a string. (Table 10)

Table 9

Operative Technique—Results

Technique	No.	Leak	Stricture	Living	Dead
Primary Retropleural	14	0	3	7	7
One layer	13	0	2A	6	7
Haight	1	0	1A	1	0
Primary Transpleural	8	3	1	2	6
One layer	2	0	1D	1	1
Haight	6	3D	0	1	5
Staged	8	0	0	3	5
Non-Operated	2	0	0	0	2
Total	32	3	4	12	20

A—Alive.
D—Dead.

Table 10

Anastomotic Complications

1. Leak—3 cases (one on POD #4, two on POD #6)
   All occurred with Primary Transpleural Height Repair
   All cases fatal. (9, 15, 37 days)
2. Stricture—4 cases—3 living, 1 dead
   Dilated X3 (2) X2 (1) X1 (2)
   Retropleural one layer (2) Haight (1)
   Transpleural one layer (1) dead

Bronchopneumonia was the major cause of death in the twenty cases which terminated fatally. (Table 11)

Table 11

Major Cause of Death (20 Cases—16 Autopsies)

Bronchopneumonia	12
Empyema	3
Pericardial effusion (Tamponade)	2
Meningitis	1
Aspiration of feedings	1
Cardiac	1
Total	20

Discussion

The most common anatomic configuration of this primitive foregut malformation, representing an incidence of 85%, is esophageal atresia with distal tracheoesophageal fistula. In perhaps 5 to 10%, there is no fistula between the distal esophagus and trachea. The other types present infrequently.

The anomaly occurs in one in 3500 births and males predominate. At least 25% are premature and the same percentage have additional critical malformations such as congenital heart disease, mongolism, hydronephrosis, absent kidney, imperforate anus, duodenal atresia, and tracheo-malacia.

Embryology

Aberrations in the development of the primary, common respiratory-digestive anlage forms the basis for this abnormality. Separation into an anterior pulmonary and posterior gut compartment by fusion of internal septae is incomplete and may produce the fistula. Intrauterine interruption of the vascular supply to the esophagus, vascular anomalies with gullet constriction, or failure of intraluminal esophageal vacuolization may explain the atresia.

Surgical Pathology

Gross anatomic information is useful in the diagnosis and management of this lesion. With the usual proximal atresia and distal fistula arrangement, the upper blind pouch is large, substantial and usually ends about 10 centimeters from the superior gum margin in the region of the azygous vein. The arterial supply from the inferior thyroid artery is rich, runs in a vertical manner, and is difficult to interrupt surgically. Conversely, the lower esophageal segment is small and flimsy, and originates from the region of the distal posterior membranous trachea, carina, or right main stem bronchus. Its arteries are distributed radially from the intercostals, and a small tracheal vessel may nourish the esophageal end of the T-E fistula. Accordingly, ischemia and necrosis of the distal esophagus is a constant hazard of the operation. Congenital stenosis and atresia below the fistula in the distal esophagus has been reported.

The attachment of the lower esophagus to the trachea in the region of the carina places this segment in some proximity to the upper pouch. In a third of such cases the two muscle walls are in contact and anastomosis is simple; in the remaining two-thirds the anastomosis is more difficult because the segments are separated by a gap varying from one millimeter to several centimeters. In the 5 to 10% of artesias without fistula, the distal esophagus is actually a small gastric diverticulum which barely extends above the diaphragmatic crura. The two segments are widely separate and cannot be joined surgically in the newborn.

Pathogenesis of Respiratory Symptoms

The respiratory distress with proximal atresia and distal fistula is instigated by three factors. Secretions collecting in the upper pouch may overflow into the trachea. Secondly, gastric juice can reflux through the tracheoesophageal fistula and inundate the lungs. Finally, the fistula provides a route for gastric distention, upward displacement of the diaphragm and interference with pulmonary function.

Diagnosis

The clinical picture begins with a history

of hydramnios in about 25% of the mothers. Profuse, bubbly oral mucous appears early and covers the baby's chin in spite of the most persistent oropharyngeal aspiration. Tachypnea and dyspnea, with intermittent episodes of choking and cyanosis soon follow. Regurgitation occurs after the initial and subsequent feedings and the aspiration exacerbates the respiratory distress. This is exaggerated in the rare atresia with proximal fistula, since the glucose water enters the respiratory tract through the fistula as well as by overflow of the proximal pouch. On physical examination, the abdomen is protuberant, flatus is obvious and consolidation may be demonstrated in the region of the right upper lobe. If the abnormality is atresia without fistula, the incidence of maternal hydramnios may be higher, pulmonary consolidation lower, and the abdomen is scaphoid. Otherwise, the findings are similar.

Roentgenograms in conventional views may show consolidation of the right upper lobe or more diffuse pneumonitis. Air in the gastrointestinal tract is seen with the usual atresia and distal fistula, although small fistulas may prevent air from leaking into the stomach. Conversely, large amounts of gastric air suggests a large fistula, and therapy becomes more urgent because of the exaggerated respiratory distress. An airless abdomen is presumptive evidence of atresia without distal fistula. On lateral chest x-rays, the proximal atretic pouch may be delineated by air but this is made clearer by the insertion of a small radio-opaque urethral catheter under fluoroscopy. Coiling of a stiff catheter at a level between the second and fourth thoracic vertebrae or demonstration of the blind pouch by one-half c.c. of contrast material concludes the regional diagnostic exercise. The contrast material should be aspirated at the conclusion of the examination.

These and other diagnostic studies are utilized to establish the presence of additional anomalies which can affect therapy. Thus, the mediastinal dissection can be done with more alacrity if a right sided aortic arch was noted preoperatively. Congenital heart, cerebral, gastrointestinal and neurologic anomalies must be considered and uncovered quickly before the correct operative approach can be planned. Associated cardiovascular anomalies are particularly lethal.

The diagnosis, then, of atresia, with fistula should be strongly suspected on the basis of (1) maternal hydramnios (2) excessive mucus, (3) respiratory distress, and (4) regurgitation. The suspicion is strengthened by the passage of a catheter into the esophagus which stops 8 to 12 cm. from the aveolar ridge. Final confirmation is obtained radiographically.

Therapy

Preoperatively, the upper pouch is aspirated intermittently through an indwelling catheter. A semi-upright position should be maintained to prevent or minimize gastric reflux through the fistula. High humidity and antibiotics are used to control pneumonia. Constant superior nursing should be started on admission and continued until the issue is no longer in doubt.

The operative management of isolated atresia without fistula has required esophageal replacement, since the two ends of the esophagus are widely separated and cannot be anastomosed under these circumstances. The operation immediately after diagnosis is gastrostomy under local anesthesia. Contrast study through the stomach with the infant in Trendelenburg position will outline the short, distal esophageal stump. Since an anastomosis at this time is technically impossible, a cervical esophagostomy is created to drain saliva and an esophageal substitute is anticipated at two years of age. The same maneuvers are in order when a large gap prevents anastomosis in the usual variety of atresia. A more recent innovation for these groups of patients includes elongation by bouginage of the proximal and distal pouches if necessary, until surgical approxi-

mation becomes feasible. (Lafer, Boley; Howard, Myers; Johnston)

Several operative approaches are available for atresia with distal fistula. The ultimate decision is based on the degree of prematurity, presence of other anomalies, pneumonia, exact anatomic configuration of the lesion, and preference of the operator. Transpleura or extrapleural ligation of the fistula and esophageal anastomosis with or without gastrostomy in one stage is ideal. This is the accepted approach in the full-term newborn with no other serious anomalies or pneumonia. Varying degrees of prematurity, pneumonitis, and concomitant abnormalities affect mortality so that staged procedures have assumed some popularity. Gastrostomy then becomes the primary, emergency step to decompress the fistula and control the pneumonia. Later, ligation of the fistula permits feeding while the upper pouch remains on suction. With perfect nursing, this arrangement is compatible with growth and development and the esophageal anastomosis can be done at a time of election. If such nursing cannot be provided, aspiration pneumonia from the upper pouch is inevitable, and in these circumstances, the very premature had best be committed to an early salivary fistula and delayed esophageal substitute. This is less than ideal but its mortality is a distinct improvement over primary repair in this group.

Postoperative Care

The acute postoperative period is critical. All the advantages of an isolette are utilized including humidification, temperature control and oxygenation. The pharynx must be carefully aspirated without injury to the fresh anastomosis. Postoperative feedings should be started with homeopathic amounts of glucose water and formula and this is initially administered by slow drip through a nasogastric or gastrostomy tube. Oral feedings are given with an 0.6 c.c. medicine dropper; this volume can be handled by the proximal esophagus and its anastomosis without overflow. (Potts)

Complications

Complications are legion but the most catastrophic is disruption of the suture line and empyema. The mortality from this can be lessened by an extrapleural approach which confines the contamination to the extrapleural space. The brassy postoperative cough is usually temporary and not related to a recurrence of the fistula, which is quite rare. Anastomotic strictures occur within three months in one-third of the survivors and are heralded by dysphagia, regurgitation, cough and recurrent pneumonia. Initial treatment consists of dilatation, started on the basis of dysphagia correlated with x-ray changes. Holinger, however, prefers to treat postoperative strictures early because of the danger of aspiration pneumonia. Resection has been advised for strictures which fail to respond to dilatation. Postoperative pneumonia can be instigated by faulty motility of the distal esophagus with reflux following vagus nerve injury.

Mortality

The overall survivors with primary definitive operations from a generous aliquot of children's and general hospitals approaches 60%; this is elevated to 70% of full-term newborns and perhaps 80% of full-term neonates who are otherwise normal. Primary anastomosis in prematures carries a 60 to 70% mortality but this can be reversed with operative staging.

Summary

The recent experience with 32 cases of esophageal artresia, with and without tracheoesophageal fistula at the Medical College of Virginia Hospitals during the past thirteen years has been reviewed and the relevant literature surveyed and discussed.

Editor's Note: A list of references may be obtained from the authors.

Permission to include patients of Drs. James Brooks and Owen Gwathmey is deeply appreciated.

Medical College of Virginia
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Surgical Treatment of Calcific Sub-Acromial Tendinitis

GEORGE A. DUNCAN, M.D.
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When conservative treatment of calcified bursitis of the shoulder fails to give relief, surgical incision and then curettage of the bursal sac offers an excellent means for cure of this condition.

THIS IS A REPORT on the surgical treatment of calcific tendinitis of the shoulder joint and a follow-up study of these cases. All of these cases have been personally operated on by me and I have also handled their post-operative treatment.

I doubt that my experiences differ from those of other orthopaedic surgeons. Either because of an increase in the incidence of calcifying tendinitis of the shoulder, or because of the increase in the population, I am seeing more cases than I did a few years ago.

Non-Operative Treatment

The basic treatment for subacromial tendinitis is conservative, or non-operative and primarily this is so for all patients. Non-operative treatment consists of several procedures, any one of which may give relief. Conservative methods will, in most patients, relieve the symptoms in 90 percent of patients. Local heat is used in its various forms to chronic conditions. In the acute cases, I believe cold applications are more efficacious than hot. Hot applications increase the blood flow to the area, thereby increasing local edema causing more pain. When the bursal sac is involved needling washing out the calcific deposit gives satisfactory results by removing the material that gives rise to the inflammatory reaction. In my hands, the administration of cortisone preparations by mouth or local injections have given only poor to fair results. The same can be said for x-ray therapy. These latter forms of treatment may give the patient temporary relief, but not a cure.

Needling and aspirating the calcific material are done at the trigger point. When needling alone is done the bursal sac is punctured and offers an opportunity for the escape of the calcium into the surrounding tissues. Frequently, Hydrocortisone is injected at the same time in hope that this will further alleviate the local inflammation.

When the calcified material is dry and granular it is almost impossible to irrigate, and the calcium deposit cannot be removed by needle.

In very acute, painful shoulders due to a calcified tendinitis, I always infiltrate the area with novocaine and xylocaine using a #27 or #24 gauge needle. With anesthesia present a #20 gauge needle is inserted at the pre-determined point of maximum tenderness. If calcium can be washed out I can expect a good result in almost all cases. If no calcium can be obtained, Hydrocortisone will be injected. In some patients with a low threshold for pain, who are having acute shoulder pain, the needling procedure can be quite painful, even though a local anesthetic is used. There may be immediate relief of pain followed by recurrence of pain with absorption of the local anesthetic. If the pain persists longer than the expected post-injection flare-up, which lasts only a

few hours and then subsides, surgical incision and curettage should then, in my opinion, be undertaken. Surgical excision of a calcified tendinitis is also advised for those patients who have had recurrent attacks of pain after various non-operative measures have failed.

Howorth stated[1] calcific deposits may be washed out or drained by multiple needle punctures or removed, by open reduction. DePaula and Kruper[2] in their excellent report on "Long Term Study of Shoulder Joints Afflicted and Treated for Calcific Tendinitis", found that conservative therapy gave good early results and residual calcium deposits were common and the long term results were not good. Surgical intervention gave better long term results in their hands.

The operation is usually done under Sodium Pentothal anesthesia, but can be done under local anesthesia. A small (1¼ to 2 inch) incision is made over the anterior aspect of the shoulder (#3 and #4). The fibers of the deltoid muscles are split then the subdeltoid bursa over the capsule is entered. The calcific mass can be brought into view by rotating the humeral head into the incision. In almost all cases, the pressure is so great the material will squirt out of the wound as soon as it is incised. The calcium is curetted out and degenerated tissue that presents itself is trimmed away. The curettage, I believe, is most important as it probably produces a scarification of the walls of the sac that causes the tissue to grow together and eliminates the cavity. I might add here that you may encounter two or three calcified deposits in one shoulder.

In my early cases, I removed the tip of the acromion and have also severed the coraco-acromial ligament. I see no reason for doing either of these procedures now and have found no difference in the end results. Also, I do not suture the small wound in the bursa; it is left open for drainage purposes. If there has been considerable inflammatory reaction in the bursa, I will inject a cortico-steroid before closure of the wound. The muscles and other layers are sutured with plain catgut, except the skin where silk or cotton is used.

In some of these shoulders, I found residual masses of cortico-steroid crystals that had previously been injected and had not been absorbed. It was present as a soft white mass which simulates a calcium mass which has ruptured.

Following the operation, the patient's arm is placed in a sling for comfort, which most patients soon discard. The day after operation they were shown dependent exercises. Most of these patients were discharged from the hospital less than twenty-four hours after their operation. It was unusual for a patient to stay over two or three days. Nine patients stayed over three days and the longest stay was six days. As their soreness subsided, the range of their exercises was increased.

In none of these wounds did any serious infection occur. In three, there was a small stitch abscess that healed after evacuation. Hematoma occurred in twelve cases and the drainage cleared up a a few days after evacuation.

Follow-up Study

From 1949 to 1961, I operated on 113 shoulders of 108 patients, as both shoulders were operated on in 5 instances.

The average age of these patients was 46.6 years. The oldest was 74 and the youngest 23 years of age. Females predominated over males in this series, 78 to 35. Also, the right shoulder predominated over the left 69 to 44. One might decide that the female is more susceptible to this condition or that they complain more and are more prone to seek medical attention. Likewise, calcifying tendinitis is thought by many people to be frequently caused by gout, but gout is not common among females. Actually, gout occurs in the female in the various series of shoulder calcifities by others reported as five to 20 percent,

whereas, in this study the females out-numbered the males two to one.

In this series of cases, I did find uric acid crystals present in two instances, but did not make a routine test of the material or a blood uric acid test. The right shoulder being the dominate one was the most susceptible in this series. If trauma is considered as a possible cause, one would think there would be more shoulders involved in men than in women.

The follow-up study was done on this group primarily by questionnaire. The response was excellent, as 83 of the 108 patients (77 percent) answered. I am sure I could not get that many private patients to give up their time and come to my office. Of the questionnaires sent out, two of these patients have died and 10 questionnaires were returned as the patients could not be located.

QUESTIONNAIRE

	Yes	*No*
1. Do you think this operation was worthwhile?	81	2
2. Do you have any pain or discomfort in this shoulder now?	11	72
3. Did you get relief after this operation?	81	2
4. Do you have any limitation of motion in your shoulder? If yes, does it interfere with your activities?	5	4
5. Is the scar unsightly or tender?	10	73
6. Would you have the operation again if needed?	80	2
7. Remarks.		

The average follow-up time in these cases was five years, the longest time being 11 years and the shortest one year.

The results of this questionnaire would indicate that this has been a successful procedure. Only two male patients out of the 83 patients operated on (excluding the two who died and the 23 who failed to answer the questionnaire) were not satisfied.

Eighty-one patients of the 83 who replied were satisfied, indicating 97 percent of favorable results. If we consider the 23 patients who failed to answer the questionnaire as complete failures, the favorable results would still number 79 percent.

In answer to question #2, 11 patients stated they did have some discomfort or pain. Most frequently their shoulders ached some with changes in the weather. Two patients stated they thought they had arthritic aching pains and not the pains of bursitis.

The answers to question #3 were the same as to #1, except that one patient qualified her answer stating that although she had immediate relief following her surgery, the pains recurred one year later and the same shoulder was re-operated in Florida. She has had no further pains.

Nine patients stated they had some limitation of motion in their shoulder, but only five stated it was sufficient to interfere with their normal activities. Two of these five cases were the failures I have reported. This loss of motion suggests that there is probably a residual arthritis present in their shoulder.

An unsightly scar was reported as being present in 10 of the 83 patients in answer to question #5. This might be eliminated by the use of a transverse incision or by more meticulous wound closures. Vertical incisions in this area always seem to have a tendency to spread.

The answers to question #6 provide a third endorsement to the worth of the operative treatment. The only negative replies were from the two failures and one questionnaire that was returned unsigned.

Most of the remarks appended to these questionnaires indicated that the operation had given these patients almost dramatic and lasting relief of their pains. Many stated they would prefer the operation to the various conservative treatments tried if they had the same type of involvement with pain in their opposite shoulder.

I am reporting my results of one form

of treatment for acute bursitis of the shoulder. Surgical ablation of the calcification is a successful procedure as these results indicate.

Discussion

There is no question in my mind, but many cases of acute, calcified bursitis of the shoulder can be relieved by conservative means. If these patients so treated fail to obtain relief or they recur, surgical incision and then curettage of the bursal sac offers an excellent means for cure of this condition. Having done 113 shoulders in 108 patients with 97 percent good results, attests to this statement.

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Clinical Center Studies of Patients Who Have Shown Unusual Host Resistance Against Cancer

The cooperation of physicians is requested in the referral of patients who have shown evidence of unusual host resistance against the growth of cancer for studies being conducted by the Surgery Branch of the National Institutes of Health in Bethesda, Maryland.

Needed for these studies are patients who have had spontaneous regression of established tumors, prolonged survival following incomplete surgical excision of their cancers, or prolonged remissions following chemotherapy. Sera from these patients will be of particular interest. Upon completion of their studies, patients will be returned to the care of the referring physician who will receive a summary of findings.

Physicians interested in having their patients considered for admission to these studies may write or telephone: Donald L. Morton, M.D., Clinical Center, Room 10-N-222, National Institutes of Health, Bethesda, Maryland 20014, Telephone: 656-4000, Ext. 63013 (Area Code 301))

Motorcycle Accidents at the University of Virginia Hospital

JAY L. ABBOTT, M.D.
Charlottesville, Virginia

There has been a tremendous increase in the number of personal injuries resulting from motorcycle accidents and this trend is likely to continue. Safety regulations and a program of safety instruction are urgently needed.

AN ASTONISHING INCREASE in the number of motorcycle accident victims arriving at our emergency room has prompted this investigation.

Method

This study is an analysis of the motorcycle accidents in the Charlottesville area during the period beginning July 1, 1964, through December 31, 1966. The patient's charts were reviewed and each patient was sent a questionnaire regarding the cyclist's experience, protective gear, attitude, etc.

Findings

There were a total of 105 accident victims treated during that period. The last six month period of the study revealed a threefold increase in the number of cases over the first six month period. This corresponded with an approximate threefold increase in the area sales of cycle registration as indicated in Fig. 1. The figures for 1966 indicate that one out of every seven registered cyclist in Charlottesville had an accident during the year. Only one rider died as a result of an accident over the three year period. Table I shows the number of various injuries sustained. Predictably, lower extremity involvement and head injuries lead the list in major injuries.

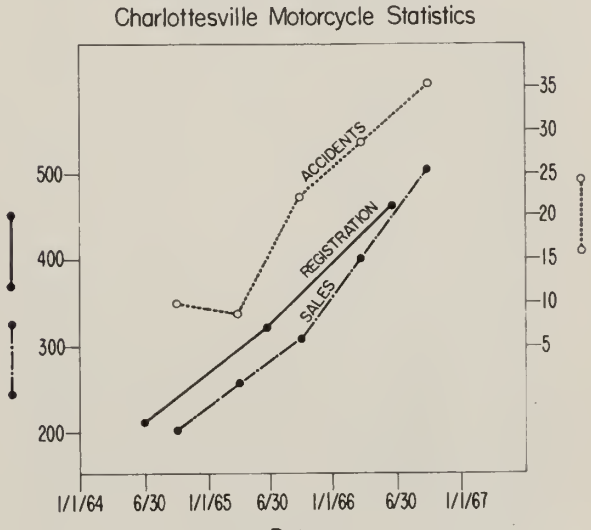


Fig. 1

Fifty-four cyclists completed and returned the questionnaire; the majority of these containing, as well, enthusiastic and intelligent comments and suggestions. Table II summarizes the questionnaire findings. It is interesting to note the high percentage of riders who were on the cycle for either the first or second time and who had not received special operating instructions. Over one half of the responders suggested helmet wearing be mandatory, yet only 28 percent were wearing them at the time of their accident. Forty percent were riding double.

Case Reports of Representative Injuries

The following cases represent the usual, yet unusual nature of motorcycle accidents.

Case No. 1. W. P., a sixteen year old high school student was driving his Yamaha on 250 By-pass when he was struck by an oncoming car. He sustained open segmental fractures of the left tibia and femur as well as abdominal trauma. He was treated for severe shock. A laparotomy and splenectomy were performed on admission. His femoral fracture healed, however, a BK amputation was necessary for treatment of a severe osteomyelitis of the tibia. He is now back playing the drums in a combo and playing golf in the 80's, but he is minus a leg and is a year and a half behind in school.

TABLE I

TYPES OF INJURIES SUSTAINED IN 105 MOTORCYCLE ACCIDENTS

Head Injury	14
Fracture of Femur	8
Fracture of Skull	7
Fracture of Tibia	7
Fracture of Clavicle	7
Fracture of Pelvis	4
Fracture of Hand	4
Fracture of Spine	3
Fracture of Facial Bones	3
Dislocation of Shoulder	3
Fracture of Ankle	2
Fracture of Forearm	2
Fracture of Ribs	2
Fracture of Humerus	2
Fracture of Scapula	2
Fracture of Olecranon	1
Fracture of Patella	1
Dislocation of Hip	1
Splenectomy	1
Orchidectomy (Testicular Contusion)	1
Minor Soft Tissue Injuries	63

TABLE II

QUESTIONNAIRE CONCERNING MOTORCYCLE ACCIDENTS

1. Was either first or second time on motorcycle	28%
2. Had *NOT* received instructions in operating motorcycle	72%
3. Were wearing a helmet	28%
4. Were wearing boots or heavy footgear	20%
5. Were wearing leather jacket	2%
6. Were riding "double"	40%
7. Were operating a borrowed motorcycle	24%
8. Were operating a small cycle (Less than 250 cc)	60%
9. Have given up motorcycling	36%

Case No. 2. F. H. is a 28 year old laborer who was driving his Harley-Davidson to work last May when he skidded on gravel and went over a bank. He sustained a rather benign appearing closed fracture of his left humeral shaft. Because of a totally functionless arm a myelogram was obtained which revealed avulsion of the C-4 and C-5 nerve roots. A year later he has still not returned to work; his arm remains totally asensory and useless and probably will come to amputation.

Case No. 3. On March 23, 1966, M. R., a 25 year old graduate student accepted a job as an aviation engineer to begin after graduation in June. Two days later, on March 26, 1966, he drove his Honda head on into a tree. One year later his bilateral femoral fractures have healed. However, his severe head injury still hampers his performing advanced problems and he is a long way from receiving his degree of aviation engineer.

Conclusions

Several facts come to light in a study of this nature. First, it can be concluded without reservation that motorcycle travel is here to stay and will continue to increase. Measures must be taken to reduce the relentless increase in serious cycle mishaps.

The most severe accidents are the result of head injuries and these usually in riders not wearing helmets. A large percentage of accidents are caused by automobile operators who do not see or recognize the motorcyclist. A high percentage of accidents are the result of inexperienced cyclists. Before conducting this investigation I prejudged the motorcycle driver as a member of an irresponsible group. From the intelligent, enthusiastic and constructive manner in which they responded to the questionnaire, I have changed my mind and feel that a more vigorous program of safety education would be well worthwhile.

Recommendations

1. Helmets must be worn while riding a motorcycle.

2. The operator, in addition to a regular driver's license, should pass a special motorcycle written and road test.
3. Riding "double" should be prohibited.
4. The wearing of a standardized bright colored fluorescent vest should be mandatory for the cycle operator.
5. Frequent periodic lectures and demonstrations conducted jointly by law officers and interested physicians regarding motorcycle safety should be carried out.

The latter recommendation should best be carried out at the local or community level. I believe a similar investigation over the next few years would then show a significant decrease in motorcycle accidents in our area.

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Let's Reminisce!

FEMALE DOCTORS—We have no opposition to suggest to the medical education of women; certainly as nurses they cannot be excelled. But when they desire to become doctors, and as such to practice we would calmly suggest that the most careful consideration be taken. The *Louisville Medical News* (May 20) well says, that "no woman has done the cause greater service than Dr. Mary Putnam Jacobi; and yet, she has given great discouragement to her medical sisters by her clear and forcible statement of the character and drawbacks of the work." Dr. Jacobi, in her recent article for the *North American Review*, says that the profession of medicine must be chosen from a strong and genuine taste, and must be adopted not from an economic pressure, but as a life work. The woman must renounce marriage for the sake of medicine, "or else there must be such an adjustment of domestic claims as shall render them and the practice of medicine by married women mutually compatible." And surely this is difficult. *Hoc est opus.* (Virginia Medical Monthly, May, 1882)

Effective Oral Diuretic Therapy Without Potassium Supplements

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IN MEDICAL PRACTICE the complaint of edema, whether "idiopathic" or secondary to numerous conditions, is one of the most frequently encountered. An effective diuretic, free of side-effects, is necessary and desirable.

Valuable as they are in diuretic therapy, the frequently used thiazides are commonly the cause of excessive potassium loss. Often the amount lost is small and causes no difficulty, but in others it has been found to produce general weakness and fatigue due to greater loss. In combination with digitalis, some patients may slip into digitalis intoxication. In those with liver disease, low potassium levels have been known to lead to hepatic coma.

To avoid such potential problems from hypokalemia, many physicians supplement thiazide diuretic therapy with potassium, either by recommending a diet rich in potassium, or prescribing potassium salts. Usually this is sufficient to avoid trouble, but not infrequently these measures may, in themselves, cause additional problems. For example, potassium salts may fail to prevent hypokalemia.[1] Recently it has been found that this medication may cause small bowel ulceration or obstruction.[2-4]

An alternate, and perhaps superior, way to avoid potassium loss would be to use a potassium-sparing diuretic, such as triamterene or spironolactone, but neither, alone, has been dramatically effective. However, these agents appear to potentiate the effect of other diuretics.[5-8] Unlike the thiazides, they retain their activity in the presence of sodium-retaining steroids. Spironolactone loses its activity unless these steroids are present, whereas triamterene retains its activity in their presence or absence.

Multiple Diuretic Therapy

This series includes 27 patients with edema (associated with various clinical conditions) treated with a combination of 50 mg. of triamterene and 25 mg. of hydrochlorothiazide. Eleven patients had not received diuretics before; sixteen were changed from other diuretics to the combination.*

The routine dosage of the combination was given once or twice a day. Periods of treatment ranged from two or three weeks to five or six months, depending on the underlying disease and its response to treatment. The average treatment period was seven weeks. In no instance was any attempt made to supplement potassium intake by medication or by diet during the study.

Results

The results of treatment with the combination of triamterene and hydrochlorothiazide are summarized in Table I. Considering both efficacy and side-effects, the overall response in the 27 patients was considered "excellent" in 22. Two of these had congestive heart-failure and required meralluride injections occasionally for control of the edema, as did one patient with "idiopathic" edema. In four patients the response was rated "good" because of lack of complete control of the edema even with concomitant therapy. One patient with chronic uremia and hypertension showed no demonstrable effect from the combination, or, for that matter, from several other diu-

*Dyazide®, Smith Kline & French.

retics tried, and his response was rated as "poor". His 1+ edema continues.

Table I also shows the causative or concurrent condition of the clinical picture of these patients. Congestive heart failure was found in five patients, obesity in five, and hypertension in five. Three patients were thought to have "idiopathic" edema, and three had diabetes mellitus. Found in two patients were the following: arthritis, hypothyroidism, asthma, emphysema, and senility. Found once were as follows: Laennec's cirrhosis, pre-menstrual edema, atrial fibrillation, reduced urinary output, fatigue, frost-bite, and chronic uremia.

Table II shows the results in 16 patients who were on other diuretics prior to this study. It shows that eleven responded better to the combination of triamterene and hydrochlorothiazide than they had responded to other diuretics and four continued to respond as well with good diuresis. Six responded better to the combination than they did to the thiazides alone.

As to side-effects, one patient noted a rash concurrent with the combination and the drug was discontinued. When the drug was resumed, no rash occurred, and the edema was reduced. Another, who was taking digitalis, complained of nausea. After stop-

Table I

Patient	No.	Age	Sex	Clinical Characteristics	Result	Comments
G.S.	1	69	M	Laennec's cirrhosis, abdominal and ankle edema	E	Definite help with edema
H.S.	2	62	F	Asthma, obesity, 2 + ankle edema	G	Rash concurrent, drug withdrawn No rash when drug restarted Slight edema remained
O.H.	3	60	F	Chronic CHF, severe; dyspnea, 4+ edema, hypertension	G	Edema reduced to 2+ BP reduced
I.G.	4	44	F	Pre-menstrual ankle edema	E	Edema controlled by one dose prn
E.S.	5	58	F	Diabetes, mild CHF, exertional dyspnea, ankle edema	E	Edema controlled
R.B.	6	58	F	Chronic atrial fibrillation; fatigue, exertional dyspnea, ankle edema	E	Edema controlled (Occasional meralluride)
E.K.	7	67	F	Ankle edema 3+, "Idiopathic"	E	Edema completely controlled
C.H.	8	51	F	Reduced urinary out-put with edema	E	Increased out-put; edema controlled maintained on one dose twice weekly
E.G.	9	68	M	Hypertensive C-V disease with mild CHF; asthma; exertional dyspnea, 3+ ankle edema	E	Nearly complete control of edema BP reduced (180/110 to 140/96)
S.C.	10	70	F	Obesity, arthritis of knees, 2+ ankle edema	E	Maintained on one dose daily
O.B.	11	59	F	Obesity, diabetes mellitus, 2+ ankle edema	G	Slight edema remaining
E.W.	12	50	F	Obesity, climacteric, abdominal and ankle edema	E	Good control, edema recurs immediately if diuretic stopped
J.K.	13	59	F	"Idiopathic" edema of ankles for 20 years, obesity	G	Incomplete control (4+ to 2+)
B.T.	14	81	F	Mild hypertension, diabetes mellitus, ankle edema	E	Occasional slight edema BP lowered
H.G.	15	94	F	Senility, hypertension, ankle edema	E	Complete control with one dose daily
F.B.	16	76	F	Rheumatoid arthritis, emphysema, 3+ ankle edema	E	Edema controlled
N.H.	17	52	F	Hypertension; facial, abdominal and ankle edema	E	Edema and BP controlled
J.G.	18	42	F	Fatigue, ankle edema	E	Edema completely controlled
E.G.	19	73	F	Chronic CHF, weakness, dyspnea, ankle edema	E	Edema controlled; occasional meralluride
A.B.	20	47	M	Post-frostbite edema of foot	E	Edema quickly controlled and maintained with one dose, prn
P.S.	21	38	F	Obesity, ankle edema	E	Edema controlled
G.T.	22	63	F	"Idiopathic edema" ankles	E	Edema completely controlled
H.T.	23	68	F	Hypothyroidism; generalized and ankle edema	E	Edema controlled well
M.C.	24	46	F	Hypothyroidism, chronic ankle edema	E	Edema controlled
M.A.	25	89	F	Severe CHF; generalized edema, emphysema	E	Edema controlled, but patient needed occasional meralluride Patient died of heart attack
R.C.	26	81	F	Senility, edema of ankles	E	Edema controlled well
J.P.	27	68	M	Hypertension, mild uremia; ankle edema	P	No effect

ping all drugs for one week, he resumed the diuretic, but not the digitalis, and had no further nausea. A third patient reported a loss of appetite, and a fourth complained of constipation and flatulence, but neither patient had to discontinue the diuretic to control these symptoms, which gradually subsided.

Serum potassium levels were checked in eight patients before the study and rechecked at various intervals throughout. Before the study, all tests were within normal limits (3.5-5.5 mEq/L), and remained so in six of the eight patients. One patient showed a decrease from 4.5 to 3.1 mEq/L after seven weeks on the diuretic combination, and the other patient showed an increase from 5.0 to 5.9 mEq/L after nine weeks. No ill effects were observed in either patient. No weakness, fatigue or other manifestations of potassium depletion were observed in any of the patients during the course of the study, including the ten patients who were treated with the combination from two to six months. No potassium supplementation was needed.

Blood urea nitrogen (BUN) levels were followed in seven patients. Of the four patients who had normal levels prior to the study, two were slightly lower, one remained the same, and one increased slightly at one month, returning to normal after two months of therapy. Three patients showed slightly elevated levels; in one it remained the same, and in two it decreased with treatment, in one to a normal level.

Changes in blood pressure were observed in 17 patients. Slight fluctuations were noted in nine normotensive patients but followed no pattern. In eight patients with mild hypertension a decrease was noted between the average pre-study reading (157/92) and the average reading during the study (142/85). No clinically severe hypertensives were included in this series.

TABLE II

Patient	No.	Previous Diuretic	Previous Diuretic	Edema Control With Triamterene and Hydrochlorothiazide
G.S.	1	Thiazides	Good	Excellent
H.S.	2	Thiazides and Reserpine	Fair	Good
I.G.	4	Thiazides	Good	Excellent
R.B.	6	Spironolactone and Thiazides—occasional meralluride	Excellent	Excellent (plus occasionally meralluride)
E.K.	7	a) Thiazides	Fair	Excellent
		b) Spironolactone and Hydrochlorothiazide	Excellent	Excellent
C.H.	8	Spironolactone and Hydrochlorothiazide	Excellent	Excellent
E.W.	12	Spironolactone and Hydrochlorothiazide	Good	Excellent
J.K.	13	Spironolactone and Hydrochlorothiazide	Good (1 or 2 a day)	Fair (two a day) Good (three a day) Very good (one a day plus Spironolactone, one a day)
H.G.	15	a) Thiazides	Fair	Excellent
		b) Spironolactone and Hydrochlorothiazide	Excellent	Excellent
F.B.	16	Benzothiadiazine	Poor	Fair (one a day) Excellent (two a day)
E.G.	19	Thiazides	Good	Excellent (occasionally meralluride)
H.T.	23	Thiazides and Reserpine	Good	Excellent
M.C.	24	Hydrochlorothiazide	Poor	Excellent
M.A.	25	Thiazides	Good	Excellent (occasionally meralluride)
R.C.	26	Triamterene	Fair	Excellent
J.P.	27	Thiazides, Resperine, Spironolactone	Poor	Poor

Case Reports

Case #1. (Patient #2) Mrs. H. S., a 62-year-old sales clerk, had been seen occasionally for five years for control of her obesity and hypertension. For 20 years previous to this she had been taking desiccated thyroid in doses up to 15 grains (one gram) daily! Edema, hypertension and asthma were all accentuated by weight gain and improved with weight loss by diet. Thiazide diuretics combined with reserpine were moderately helpful. For two years prior to the study she was seen only occasionally for acute asthmatic attacks. She had gained weight to her maximum of 225½ lbs. and

had pitting 3+ ankle edema. There was slight asthma and only a moderate elevation of blood pressure. Given two tablets of the combination diuretic a day, with a low calorie diet, she lost 8½ lbs. in one week. The patient discontinued the medication for one week because of a generalized rash, and promptly gained weight and developed edema again. When the medication was again started, there was no recurrence of the rash, her edema and blood pressure were controlled, and she gradually lost 25 pounds. The combination diuretic was continued even through a period of cardiac insufficiency with angina, and was still effective.

Case #2. (Patient #12) Mrs. E. W., 49, obese due to nervous over-eating, menopausal, somewhat hypothyroid, had complained of ankle and abdominal edema for years. At first it was premenstrual, and she had been treated with thiazides alone and later with additional spiralactone; but with the climacteric the edema had become constant, and at the time of the study she was requiring daily medication for control. It was found that two doses of the combination diuretic daily controlled the edema completely, and more effectively than two doses of the thiazide-spironolactone combination.

Case #3. (Patient #6) Mrs. R. B., 58, saw her son struck and killed by an automobile 25 years previously and credited the shock with being the cause of her auricular fibrillation. Edema has increased over the years and has been accompanied by many symptoms such as anxiety, sensations of smothering, weight gain of six or more pounds in a day's time, abdominal fullness, and shortness of breath. Only modest cardiomegaly and an unremarkable ECG were found; there was no indication of liver or renal disease; she was diagnosed as the so-called "idiopathic" edema syndrome. Elective surgery for perineal repair was deleted because of her edema and nervousness. She had been found to have some relative hypoglycemia, and this was helped by diet. Unable to tolerate whole-leaf digitalis, she was given digitalis glycosides. Numerous diuretics were tried. The thiazides alone were ineffectual; combined with adequate doses of spironolactone the edema was well-controlled most of the time, but an occasional dose of meralluride was found necessary. The combination diuretic was equally effective. At times she needed spironolactone, triamterene and hydrochlorothiazide, plus an injection of meralluride, for episodes of severe edema. Usually smooth maintenance was accomplished by the combination diuretic.

Summary

Twenty-seven edematous patients with various diseases including congestive heart failure, mild hypertension and diabetes were treated with a combination of triamterene (50 mg.) and hydrochlorothiazide (25 mg.) and 22 (81.5%) obtained complete control of edema. Four patients had incomplete but "good" control. Eleven of 16 patients responded better to the combination than they had to other diuretics. On eight patients potassium levels and on seven BUN levels were taken before, during and after treatment, and these levels were practically unaffected. None of the patients treated showed symptoms of potassium depletion, and no supplementary potassium was administered. It is felt that this combination diuretic is safe and effective for frequently encountered and troublesome edema.

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The Future of Blood Therapy

The time may come when the first "deposit" of blood for a person's account in a long-term blood bank will be taken from the placenta before his birth.

Later in life, he could begin making deposits in the bone marrow bank. This super-cold storage area would preserve supplies of his bone marrow in case he needed it for a bone transplant, or to replace marrow damaged during heavy radiation therapy.

Are these far-fetched, science-fiction ideas? Perhaps, but not necessarily, says pathologist Israel Davidsohn, M.D., in the December 4th Journal of the American Medical Association. As part of the Journal's special section on blood therapy, Dr. Davidsohn contributed ideas on what tomorrow's therapy may consist of, based on the present rate of progress.

"Good methods for marrow preservation are already available. Present accomplishments justify the hope that, with further improvement and simplification of equipment and methodology, red cells and marrow may be preserved indefinitely. . . ."

A main purpose of these long-term blood and marrow banks would be to end the problems of proper matching, sensitizing reactions, and occasional hepatitis infections that are a part of current blood therapy. Long-term storage and periodic deposits would ensure everyone all the blood he needs when he needs it. In fact, there is evidence that frozen blood loses some of the characteristics that cause immune reactions when placed in a patient's body.

"Advances already achieved . . . justify this hope. Storing the blood would not require much more space than safety deposit boxes in banks."

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Aplastic Anemia Following Chloramphenicol Therapy

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Chloramphenicol has been implicated in another case of aplastic anemia. The possibility of inducing a serious blood dyscrasia must be considered when using this drug, especially if large doses are given.

APLASTIC ANEMIA is the most important of the drug induced blood dyscrasia because of the high mortality estimated at 50-80% and because this represents approximately one-third of all reported blood dyscrasia associated with drug reactions. Chloramphenicol is the drug most often associated with aplastic anemia although the incidence of aplastic anemia in patients treated with chloramphenicol has been estimated to be as low as 1 in 800,000. There is no test which provides proof of an etiological relationship. Conclusions must be drawn on the basis of statistics and personal judgment.

Report of Case

R. M., an 18 year old colored male, was first admitted to the hospital July 24, 1965, through September 11, 1965, for fractures of the right femur, frontal sinus, nasal bones, right and left orbits, right zygomatic arch, and medial wall of the maxillary antrum sustained in an automobile accident. Initial blood studies revealed a hemoglobin of 14.2 grams percent; hematocrit 43 percent, white cell count 32,000 with 76 percent polymorphonuclear cells, 14 percent stabs and 10 percent lymphocytes.

He was placed on prophylactic penicillin and streptomycin, and after his condition stabilized, he was taken to the operating room for repair of his fractures. On the day of surgery, his hemoglobin was 13.8 grams percent; hematocrit 44 percent, white cell count 13,450 with 75 percent polymorphonuclear, 45 percent stabs, 21 percent lymphocytes and platelets were reported as adequate. Following surgery, he was placed on chloramphenicol 500 milligrams every six hours. His postoperative course was uneventful, and he was discharged 9/11/65 in a full body spica cast to be followed as an outpatient. Hemoglobin 13.8 grams percent; 6 mononuclear, platelets 228,000 on the day of discharge. He failed to keep his return appointment and was not seen until October 20, 1965, when he returned to the outpatient department to have the wires removed from his jaw. He returned to the Emergency Room that evening in acute distress, bleeding readily from the oral cavity. Other pertinent physical findings were BP 130/40, Pulse 130/min., Respirations 20/min., and shallow. The conjunctiva were pale. No enlarged lymph nodes were palpable, and the abdominal examination was negative.

Pertinent laboratory data was as follows: Hemoglobin 2.2 grams percent, hematocrit 6, platelets 40,000, white blood count 2,200 with 23 polymorphonuclear, 73 percent lymphs, 4 percent monocytes, Prothrombin consumption times 33 seconds, prothrombin time 16 seconds (control 13 seconds), LE prep and heterophil tests were negative,

total protein 6.6 grams with 4.2 grams albumin, 2.4 grams globulin. Bone marrow aspiration showed moderately hypocellularity and no megakaryocytes. Marked myeloid hypoplasia with most of the myeloid elements being promyelocytes and myelocytes. The erythroid elements were relatively if not absolutely increased with a shift to the earlier forms.

The patient was tranfused with three units of whole blood and started on prednisone 40 milligrams every hour. He was also given testosterone enanthate 600 milligrams intramuscular, October 24, 31 and November 7 and 13.

The hospital course was uneventful on the above treatment except for the appearance of several small scattered retinal hemorrhages. Prednisone was gradually tapered and periodic blood examinations revealed November 1, hemoglobin 10.8 grams percent, hematocrit 31, platelet count 10,000, white blood count 3350, reticulocyte count 2.4 percent; November 7, hemoglobin 12.0 grams percent, hematocrit 34, platelet count 13,000, white blood count 4350, reticulocyte count 1.7 percent; November 13, hemoglobin 12.2 grams percent, hematocrit 36, platelet count 20,000, white blood count 4500, and reticulocyte count 3.0 percent and April 19, 1966, white blood count 5600, platelet count 176,200.

Comment

The exact cause of chloramphenicol induced toxicity is unknown. Reports of an increased incidence in children, a female to male ratio of 3:1[2] and reaction after a short course of therapy[3] may be used to support the thought of specific host factors such as hypersensitivity. There are other studies, however, which may be indicative of a dose relationship.[4-5] In one of these,[5] progressive erythroid depression mild thrombocytopenia, and evidence of disturbed granulocytic production occurred in patients given six grams of chloramphenicol per day for two weeks or more. These changes promptly disappeared after the drug was stopped. Bone marrow depression was noted to occur regularly with plasma chloramphenicol levels of 25 micrograms per cc or more. Hematological abnormalities in a group of patients that received two grams of chloramphenicol per day did not differ significantly from that in a control group except in two cases of bone marrow depression which cleared spontaneously when the drug was stopped. This suggests that toxic bone marrow depression is a dose related pharmacologic property of chloramphenicol.

Treatment of aplastic anemia is supportive and consists of blood transfusions to maintain a hemoglobin at which the patient is comfortable; steroids in an attempt to improve the leukopenia and thrombocytopenia; early recognition and treatment of infections. However, testosterone has been added in the last ten years with the favorable reports by Shakidi and Diamond.[6-7] Their rationale for using testosterone was based on a paper published by Kennedy and Nathanson.[8] These two investigators found that a substantial number of patients with carcinoma of the breast treated with testosterone had a rise in red blood cell count with an accompanied rise of the hematocrit and hemoglobin, and bone marrow examination revealed euthyroid hyperplasia although the mechanism of hemapoietic stimulus by androgens is unexplained.

Summary

A case of successfully treated chloramphenicol induced aplastic anemia is reported with brief comments concerning the present mode of treatment and possible etiological factors.

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Clinical Center Study of Patients with Angina Pectoris

The cooperation of physicians is requested in the referral of patients for a study of the most recent advances in treatment of patients with angina pectoris, being conducted by the National Heart Institute at the Clinical Center, National Institutes of Health, in Bethesda, Maryland.

Patients under 60 years of age with severe angina pectoris will be admitted for intensive study to evaluate the severity of the disease and to determine the effectiveness of optimal medical management. Operative intervention will be offered to those patients who are felt to be suitable candidates. Subsequently, follow-up studies will be performed to determine the degree of improvement brought about by these procedures. Upon completion of their studies, patients will be returned to the care of the referring physician who will receive a summary of findings.

Physicians interested in having their patients considered for admission to the Clinical Center for this study may write or telephone: Eugene Braunwald, M.D., Clinical Center, Room 7-N-220, National Institutes of Health, Bethesda, Maryland 20014, Telephone: 656-4000, Ext. 65095 (Area Code 301)

Cylindroma of Cervical Trachea

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Tumors of the trachea are rare, especially the cylindroma. The diagnosis and management are discussed.

BECAUSE of the rarity of tracheal tumors and the recent pre-operative diagnosis of a cylindroma of the cervical trachea, this case report is presented.

Case History

The patient, admitted 12/12/66, is a 50 year old white female who entered the hospital with the chief complaints of choking sensation and noisy respiration of approximately two months duration. Her respiratory difficulty was intermittent and with no particular relationship to activity. She had no cough, dysphagia, or hemoptysis.

A goitre was present for many years. There were no signs of recent increase in size or functional alteration.

The past history was non-contributory. There was no history of trauma, allergy, metabolic or infectious diseases.

On physical examination her color was good and appearance healthy. The blood pressure, respiratory rate and pulse were all within limits of normal. There was, however, a definite respiratory stridor. Both thyroid lobes were moderately enlarged and no nodes palpated.

The lungs were clear except for noisy respiration. The other systems were normal.

The routine laboratory findings were normal. A radioactive iodine uptake study was normal.

The vocal cords moved well on indirect laryngoscopy. No pathology seen above the cords.

Cardiopulmonary functional studies suggested an upper respiratory block.

X-ray Findings

12/12/66 Films of the chest (Fig. 1) showed clear lungs with no obstructive emphysema or atelectasis. Trachea in the mid-



Fig. 1. Chest. The lungs are clear. No obstructive emphysema.

line. A lordotic view confirmed the midline position of the trachea. An esophagram (Fig. 2) was negative. The swallowing act was normal. The esophagus was not displaced and no intrinsic lesion seen.

12/13/66 On a lateral view of the neck

(Fig. 3) a semi-spherical defect was seen within the air column of the trachea just below the ventricle of the larynx.



Fig. 2. Esophagram. No primary lesion. No extrinsic pressure or displacement.

Tomography was performed the same day.

Tomograms (Figs. 4 & 5) of the larynx and upper trachea confirmed the presence of a mass measuring three centimeters in diameter. The mass arose mainly from the posterior and left lateral walls of the trachea. The mass was smooth and sharp in outline. The airway was almost compromised.

The preoperative diagnosis was cylindroma or squamous cell carcinoma.



Fig. 3. Lateral neck. Below the larynx a smooth defect (arrow) within the posterior part of trachea.

Following tomography, and because of increasing stridor, a low cervical tracheotomy was performed.

12/17/66 chest and lateral neck (Fig. 6).

A tracheotomy tube was seen in place. The lungs showed good aeration. There was moderate subcutaneous emphysema in the neck. The defect in the trachea representing tumor was again noted.

The patient's condition improved and an elective endoscopic resection of the tumor carried out under general anesthesia. The base of the tumor was fulgurated.

The tissue report was cylindroma of trachea.

Discussion

Tumors of the trachea are relatively rare. The ratio of laryngeal tumors to tracheal tumors ranges from 300:1 to 800:1 (Seamen). For every primary tumor of the trachea one might expect to find between 300 to 400 bronchial carcinomas (Fleming, 1962).



Fig. 4. Tomogram. The mass is well delineated (arrow). Mass almost fills lumen of trachea.

Few tracheal tumors have been diagnosed before death.

Benign tracheal tumors (adenoma, fibroma, papilloma, hemangioma, hamartoma and leiomyoma) are quite rare. The malignant tumors include squamous cell carcinoma, adenocarcinoma and cylindromas.

The secondary tumors of the trachea (Sautter, 1960) originate from the thyroid, especially the anaplastic type which go through all layers of the trachea. The thyroid gland is generally markedly enlarged. Carcinoma of the esophagus by this time presents with dysphagia.

The cylindromas are a specific type of



Fig. 5. Lateral neck following tracheotomy. Defect or mass (arrow) persists.

adenocarcinoma, characterized by well differentiated cords of cells and found in the salivary glands and upper respiratory tract. Previous publications have referred to the cylindromas as adenoid cystic carcinoma, adenocystic basal cell carcinoma, and malignant adenoma (Fleming, 1962).

Cylindromas spread mainly by local invasion. Spread to distant lymph glands by the lymphatics is rare. Hematogenous spread is also rare.

Significantly, these tumors have a long

natural history, frequent local recurrences and pulmonary metastases.

Several large series of primary tumors of trachea and bronchus were reviewed. Markel (1964) and his group did not present any cases of carcinoids or epidermoid carcinomas of the trachea. They and others (Fleming, 1962) doubt the existence of tracheal carcinoids. From 1934 to 1962 they collected 87 cases of cylindromas and only two of these tumors were located in the upper trachea below the larynx.

Fleming (et al. 1962) presented four tracheal tumors. Three of the four cases were cylindromas. Two of their cases were located just above the carina. The only case of squamous cell carcinoma presented just below the larynx.

The cylindromas (Soutter, 1960) show an equal sex distribution and generally occur in the 30-50 year old age group. Most cylindromas extend beyond the cartilaginous wall of the trachea or into the surrounding tissue. They may protrude into the lumen or remain beneath the mucous membrane. Growth is slow. This group had one cylindroma in cervical trachea which invaded the thyroid gland and caused cord paralysis.

Soutter and his group (1954) reported 56 carcinoids and four cylindromas collected in a period from 1909 to 1954. All the carcinoids arose in bronchi. Three of the cylindromas were in the trachea.

The above series (Wilkins Jr., 1963) was extended to 1963. From 1909 to 1963 a total of 69 carcinoids, seven cylindromas, and six epidermoid carcinomas were collected. In the entire series a diagnosis was established by x-ray only six times.

In another recent series (Fleming, 1962) the diagnoses of cylindromas were initially made at autopsy and bronchoscopy. In retrospect the tumor masses were seen on plain film study of chest, special oblique bucky examination, and tomography.

A history of dyspnea, cough, wheeze or stridor should provide the clues of airway block. Stridor should make one very suspicious of a block higher in the airway-trachea.

Chest x-rays taken in expiration and inspiration along with a lordotic view are recommended. They are usually initially negative for tracheal lesions. Fortuitously one might pick up a defect within the trachea on a lordotic view.

The lateral view of the neck and bucky obliques of the thoracic inlet, plus the presenting sign of stridor, in our patient were most suggestive of the diagnosis. Tomography which followed was confirmatory. The smooth defect within the cervical trachea was almost conclusive of a cylindroma or squamous cell carcinoma.

In other series, bronchoscopy, along with biopsy, usually established the diagnosis. However, in one case report of a cylindroma of the lower trachea (Kleinman, 1959), two bronchoscopies were negative. The diagnosis was made by tomography. Bronchoscopy was also reported negative on several occasions especially in regard to proximal tracheal lesions near the larynx (Fleming, 1962).

Since our patient's airway was already compromised, and the location of the tumor was demonstrated by tomography, no further diagnostic workup was deemed necessary, such as bronchoscopy or tracheography.

Low cervical tracheostomy was followed by endoscopic resection of the tumor.

The patient was discharged from the hospital several weeks later in good condition.

Because cylindromas are very slow growing and mainly locally invasive, we hope the above conservative management will prove to be successful for many years. Cylindromas do tend to recur. However, they have been successfully treated by local resection (DeBakey, 1964-5), and the development of tracheoplastic operations have made extensive tracheal resections possible (Clagett, 1952). Radiation therapy has also been successful (Tinney, 1945; Fleming, 1962). Good palliative benefits for inoperable tra-

cheal cylindromas can be expected by radiation (Vieta, 1957).

Summary

Tracheal tumors are rare. However, these tumors, especially cylindromas, should be considered in any case presenting with upper respiratory obstructive symptoms.

The response of cylindromas to treatment (surgery and radiation) has been briefly discussed.

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C. & O. Hospital
Clifton Forge, Virginia

Noise in the Home

That roaring vacuum cleaner, grumbling garbage disposal, and booming hi-fi set in your home may be hazardous to your health, suggests a report in the October 16th Journal of the American Medical Association.

The collection of noisy appliances and electronic "status symbols" raises sound levels in some homes to disagreeable intensity, writes Lee E. Farr, M.D., a Houston physician who has long been concerned about the effects of noise on health. "Home sounds can threaten the health and well-being of one's emotional state. Exposure to noise . . . may lead to outbursts of fury or threats, neither of which satisfies, and each of which leaves frustration as a legacy."

Continuous loud noise can damage hearing, but this isn't the main problem in the home. Too much noise simply upsets people, particularly those who are already suffering from illness, such as the so-called tension ills, duodenal ulcer, anxieties, and emotional problems.

The kitchen is particularly faulty. It simulates "an old-fashioned boiler factory. The ventilating fan over the stove, the dishwasher, the garbage disposal unit, the blender, all make significant contributions to sound which the steel cabinets and hard plastic surfaces reflect and augment."

"It is time that man realizes that his home can be designed to acoustic criteria, resulting in a pleasant environment for him and medically conducive to a state of well-being."

Dr. Farr is on the staff of the Department of Medicine, University of Texas, M.D. Anderson Hospital and Tumor Institute, Houston.



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There are more than 30 million people in America who are overweight. During the next year, you probably will see more than 1,000 of them in your own practice.

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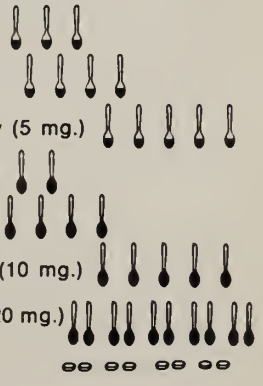
Precautions: Lomotil is a Federally exempt narcotic preparation of very low addictive potential. Recommended dosages should not be exceeded, and medication should be kept out of reach of children. Should accidental overdosage occur signs may include severe respiratory depression, flushing, lethargy or coma, hypotonic reflexes, nystagmus, pinpoint pupils, tachycardia; continuous observation is recommended. Lomotil should be used with caution in patients with impaired liver function or those taking addicting drugs or barbiturates.

Side Effects: Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness, insomnia, numbness of the extremities, headache, blurring of vision, swelling of the gums, euphoria, depression and general malaise.

Dosage: The recommended initial daily dosages, given in divided doses until diarrhea is controlled, are:

Children: Total Daily Dosage

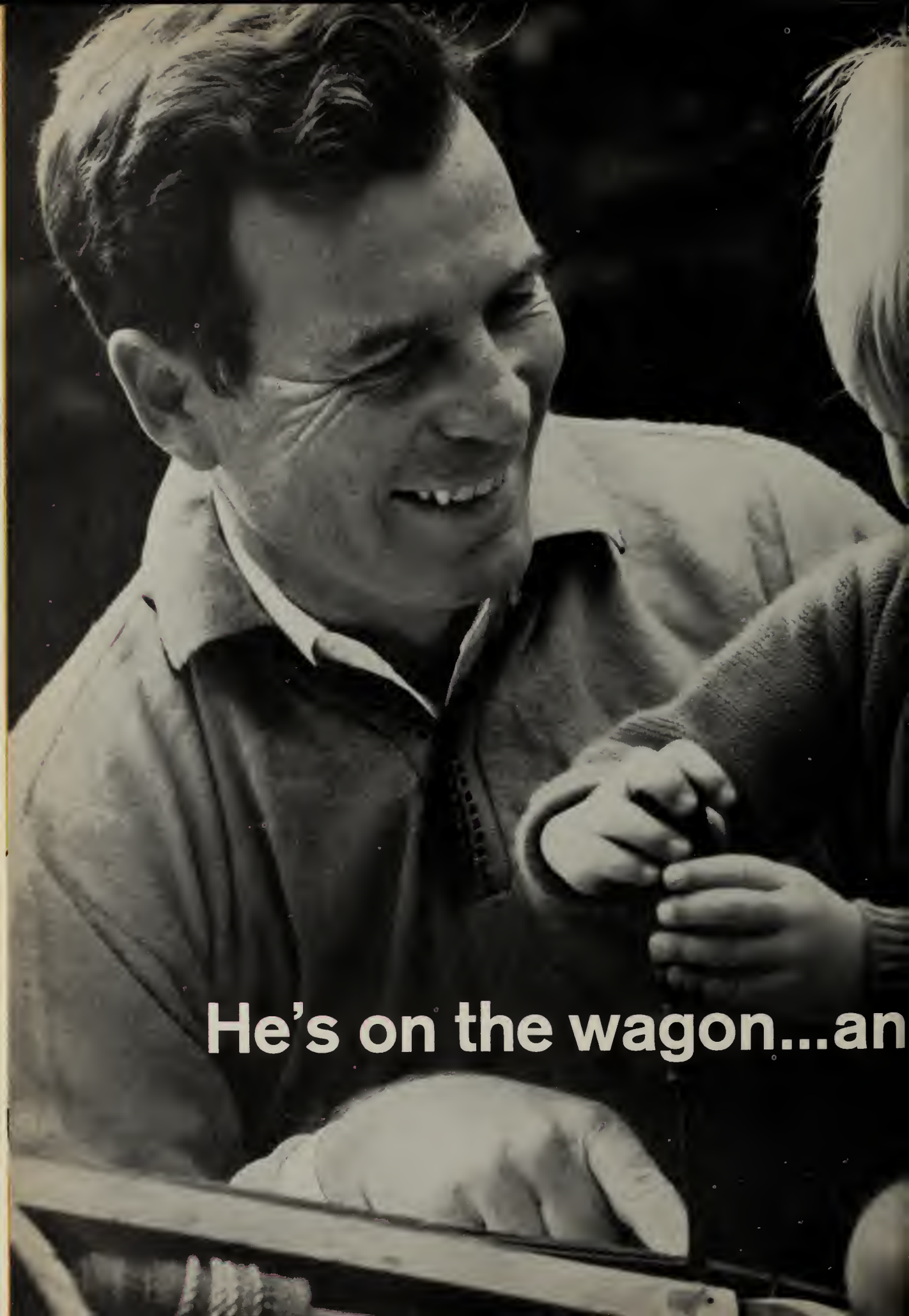
3-6 mo. . .½ tsp.* t.i.d. (3 mg.)
6-12 mo. .½ tsp. q.i.d. (4 mg.)
1-2 yr. . . .½ tsp. 5 times daily (5 mg.)
2-5 yr. . . .1 tsp. t.i.d. (6 mg.)
5-8 yr. . . .1 tsp. q.i.d. (8 mg.)
8-12 yr. . .1 tsp. 5 times daily (10 mg.)
Adults: . .2 tsp. 5 times daily (20 mg.)
or 2 tablets q.i.d.



*Based on 4 cc. per teaspoonful.

Maintenance dosage may be as low as one-fourth the initial daily dosage.

SEARLE *Research in the Service of Medicine*


He's on the wagon...an

How well does Vistaril relieve the symptoms that plague an alcoholic during the recovery period? Doctors Knott and Beard of the Alcoholic Rehabilitation Unit, Tennessee Psychiatric Hospital and Institute, recently conducted a double-blind study comparing Vistaril and another well-stablished antianxiety agent with placebo in 60 chronic alcoholic patients.[1]

The investigators conclude: "It was the opinion of the staff that hydroxyzine was generally more effective than chlordiazepoxide, for the following reasons: hydroxyzine was equally if not more effective in reducing anxiety and tension and it produced less daytime sedation...."[1] (See results on succeeding pages.)

Here is new evidence that Vistaril can ease tension, allay anxiety in chronic, hospitalized alcoholic patients. *But you might also choose Vistaril for what it doesn't do.* Although not evaluated in this study, Vistaril is reported to be non-euphorant, and its low toxicity makes it relatively safe. Best of all, Vistaril is non-habituating. To date, after more than ten years of clinical use, there have been no reports of dependency in patients receiving Vistaril.

With Vistaril, it is as easy to stop therapy as it is to start.

the study:[1] Sixty chronic alcoholic patients were hospitalized and randomly assigned to one of three oral double-blind treatment regimens for three weeks. Twenty patients received hydroxyzine (Vistaril®), 100 mg. q.i.d.; twenty others were given chlordiazepoxide, 25 mg. q.i.d.; and the remaining twenty received placebo capsules q.i.d. The capsule code was not made known until after completion of the study and analysis of the data. Response was measured with a modification of the Brief Psychiatric Rating Scale as originally outlined by Overall and Gorham.* Subjects were rated daily by trained staff members.

SYMPTOM RATING KEY

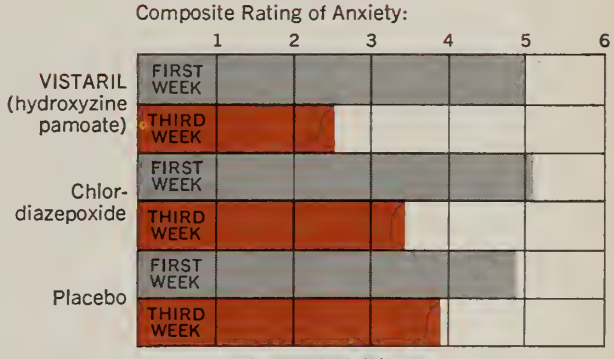
1=Not Present	4=Moderately Severe
2=Mild	5=Severe
3=Moderate	6=Extremely Severe

The figures which are given below represent the composite conclusions of the staff based on daily ratings during initial and final weeks of the study.

*Psychological Reports 10:799, 1962

the results[1] DURING FIRST WEEK DURING THIRD WEEK

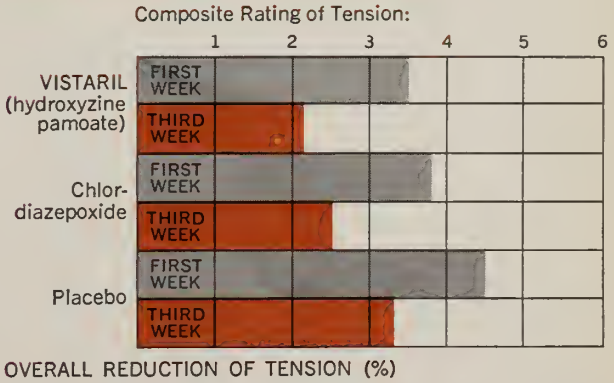
Anxiety reduced with Vistaril



OVERALL REDUCTION OF ANXIETY (%)

VISTARIL (hydroxyzine pamoate)	Chlordiazepoxide	Placebo
50%	33%	20%

Tension eased with Vistaril



OVERALL REDUCTION OF TENSION (%)

VISTARIL (hydroxyzine pamoate)	Chlordiazepoxide	Placebo
40%	34%	27%

Vistaril®
HYDROXYZINE PAMOATE)

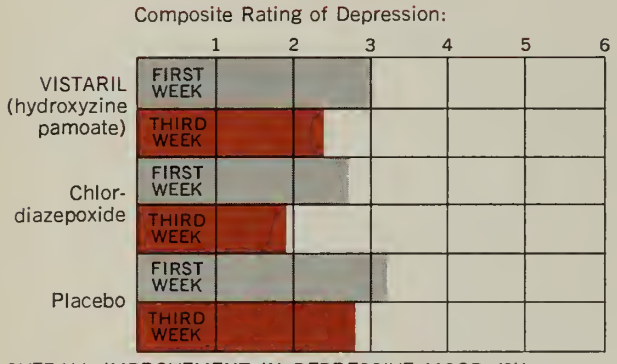
PLEASE SEE LAST PAGE FOR PRESCRIBING INFORMATION

In Alcoholism...

"Both hydroxyzine and chlordiazepoxide were generally more effective than the placebo. In some aspects, hydroxyzine was superior to chlordiazepoxide, which is currently the most frequently used psychotropic drug in the management of alcoholism."[1]

DURING FIRST WEEK DURING THIRD WEEK

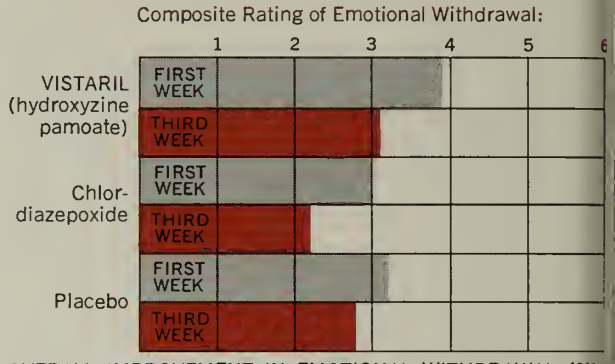
Depressive mood improved with chlordiazepoxide



OVERALL IMPROVEMENT IN DEPRESSIVE MOOD (%)

VISTARIL (hydroxyzine pamoate) 20% | Chlordiazepoxide 30% | Placebo 13%

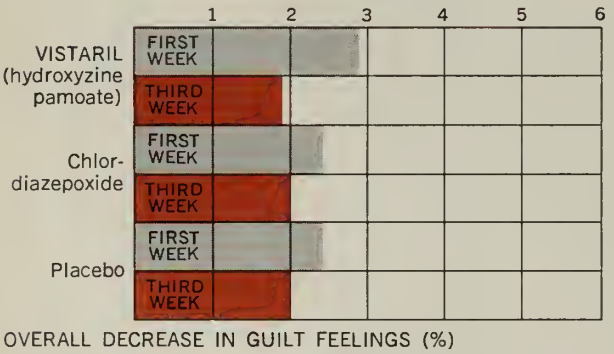
Emotional withdrawal counteracted with chlordiazepoxide



OVERALL IMPROVEMENT IN EMOTIONAL WITHDRAWAL (%)

VISTARIL (hydroxyzine pamoate) 21% | Chlordiazepoxide 27% | Placebo 13%

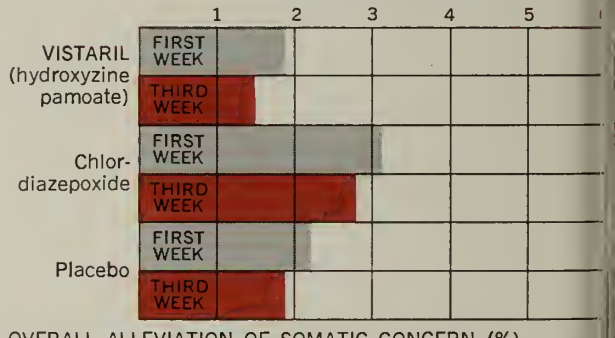
Guilt feelings allayed with Vistaril



OVERALL DECREASE IN GUILT FEELINGS (%)

VISTARIL (hydroxyzine pamoate) 34% | Chlordiazepoxide 17% | Placebo 17%

Somatic concern alleviated to minor degree in each group



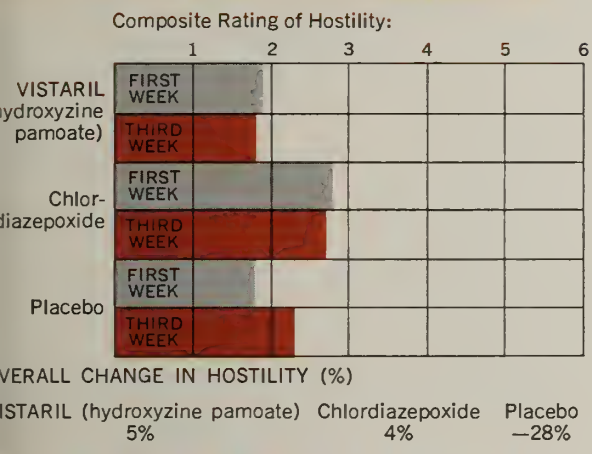
OVERALL ALLEVIATION OF SOMATIC CONCERN (%)

VISTARIL (hydroxyzine pamoate) 17% | Chlordiazepoxide 13% | Placebo 14%

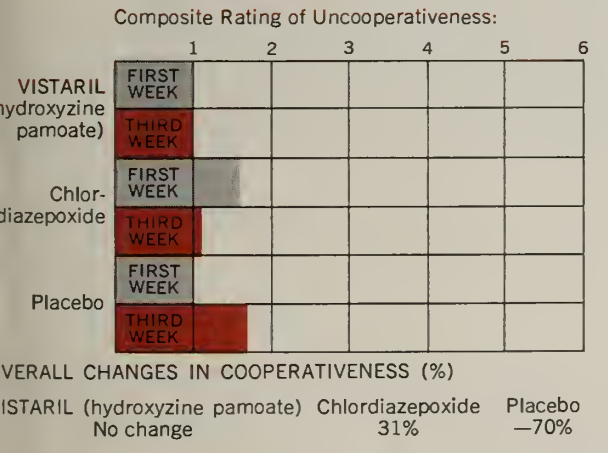
Less drowsiness with Vistaril

	Drowsiness	Dizziness	Mouth Dryness	Increased Motor Activity	Ataxia	Nausea	Comments
VISTARIL (hydroxyzine pamoate)	6 patients (mild)	3 patients (mild)	2 patients (moderate)	—	—	—	Side Effects Not Treated
Chlordiazepoxide	10 patients (severe in 6)	4 patients (mild)	—	1 patient (moderately severe in first week but gradually subsided)	—	—	Side Effects Not Treated
Placebo	2 patients (mild)	2 patients (mild)	—	—	1 patient (mild)	2 patients (mild)	Side Effects Not Treated

Increase in hostility minimized with Vistaril and chlordiazepoxide



Cooperativeness not a significant problem



n Alcoholism...

Vistaril®

HYDROXYZINE PAMOATE)

BRIEF SUMMARY

Contraindications: Hypersensitivity to hydroxyzine. The parenteral solution, for intramuscular or intravenous use, *must not* be injected subcutaneously or intra-arterially.

Hydroxyzine, when administered to the pregnant mouse, rat, and rabbit induced fetal abnormalities in the rat at doses substantially above the human therapeutic range. Clinical data in human beings are inadequate. Until adequate data are available to establish safety in early pregnancy, hydroxyzine is contraindicated during this period.

Precautions: Hydroxyzine may potentiate the action of central nervous system depressants such as narcotics and barbiturates. In conjunctive use, dosage for these drugs should be decreased, as much as 50%. Because drowsiness may occur, patients should be cautioned against driving a car or operating dangerous machinery. The usual precautions for intramuscular injection should be followed; soft-tissue reactions have rarely been reported when proper technique has been used. Hydroxyzine parenteral solution for intramuscular use should be injected well within the body of a relatively large muscle. In adults, the preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus), or the mid-lateral thigh. In children, preferably the mid-lateral muscle of the thigh. In infants and small children the upper outer quadrant of the gluteal region should only be used when necessary, as in burn patients, in order to minimize the possibility of damage to the sciatic nerve. The deltoid area should be used only if well developed, such as in certain adults and older children, and only with caution to avoid radial nerve injury. Injections should not be made in the lower and middle thirds of the upper arm. Aspiration should be done to help avoid intravascular injection. On reported intravenous injection a few instances of digital gangrene have occurred distal to the injection site, considered to be due to inadvertent intra-arterial injection or possibly periarterial extravasation. Therefore, particular caution (aspiration and site injection) should be observed to insure injection only into intact veins; avoid either intra-arterial injection or extravasation. Intravenous administration should be accomplished slowly, no faster than 25 mg. per minute, and not to exceed 100 mg. in any single dose. In order to avoid possible adverse effects it is recommended that hydroxyzine parenteral solution be diluted to at least 50 cc. with sterile normal saline and administered over a period of four minutes or more, preferably into the tubing of a running intravenous infusion.

Adverse Reactions: Drowsiness may occur; if so, it is usually transitory and may disappear in a few days of continued therapy or upon dosage reduction. Dryness of the mouth may occur with higher doses. Involuntary motor activity, including rare instances of tremor and convulsions, has been reported, usually with higher than recommended dosage.

When this product is given intravenously undiluted, minimal amounts of intravascular hemolysis occur at the site of injection. Giving the maximum recommended intravenous dose (100 mg.) to adults results in immediate transient hemolysis with the liberation of a total of 2-3 grams of hemoglobin, which, in some individuals, can cause small amounts of hemoglobinuria. This compares with the normal red cell destruction from which approximately 8 Gm. of hemoglobin are liberated every 24 hours. If the hydroxyzine is diluted with 50 cc. of normal saline and given during a period of four minutes or more, this phenomenon does not occur.

Supply: Vistaril (hydroxyzine pamoate) Capsules: Equivalent to 25 mg., 50 mg., 100 mg. hydroxyzine HCl. Vistaril (hydroxyzine pamoate) Oral Suspension: Equivalent to 25 mg. hydroxyzine HCl per 5 cc. teaspoonful. Vistaril (hydroxyzine HCl) Parenteral Solution: 25 mg./cc.—10 cc. vial and 50 mg./cc.—2 cc. and 10 cc. vial; Isoject,® 25 and 50 mg. per cc., 1 cc. per unit.

More detailed professional information available on request.

Reference: 1. Knott, D.H. and Beard, J.D.: GP 36:118, September, 1967.

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References: 1. Shuman, C.: Am. J. Med. Sci., 225:54, 1953. 2. Perchuk, E., et al.: Angiology, 12:102, 1961. 3. Rawls, W., et al.: Med. Times, 87:818, 1959.

6/67 Q-706A

Clinicopathological Conference . . . .

Recurrent Neck Mass Following Irradiation of Hodgkin's Disease

Prepared and Edited by

JOHN H. MOON, M.D.
PAGE HUDSON, M.D.
Medical College of Virginia

DISCUSSANTS:

Kinloch Nelson, M.D., Dean, School of Medicine; Professor, Department of Medicine

Fairfield Goodale, M.D., Professor and Chairman, Department of Pathology

Clinical History

This 25 year old white female was seen in November of 1965 because of a lump in the region of her larynx which had been present for about three months and was becoming more prominent. It had caused no difficulty in swallowing or phonation and there had been no local tenderness or increased heat.

The patient had a similar lesion in her right neck in 1959. A thyroid neoplasm was suspected. Exploration of her neck revealed that the nodule was a lymph node. It and several adjacent nodes were removed. The thyroid appeared normal. Tissue diagnosis on the nodes was "Hodgkin's Disease". She was treated with radiation therapy to the right supraclavicular region with 3000 r in air with complete regression of her disease and was subsequently followed at regular intervals with no evidence of recurrence until January of 1962 at which time she developed two small lymph nodes in the right side of her neck. A right radical neck dissection was performed which showed "Hodgkin's Granuloma" in four lymph nodes which had been removed. She received no further therapy until May of 1962, at which time she developed lymph node enlargement in the left axilla and was given radiation therapy of 3000 r (depth dose) to the left supraclavicular and left axillary areas over a period of 22 days, with regression of the lymphadenopathy. She developed mediastinal involvement in February of 1963 and received 3800 r (depth dose) over a 24 days period without difficulty and with clearing of her chest films. She then remained well until the onset of her present illness in August, 1965.

Review of systems was essentially negative. Family history was unremarkable. Past medical history was negative except as noted in her present illness.

Physical examination: BP 124/76, P. 108, R. 20, T. 99.2°, weight 144 pounds. Mucous membranes were of good color. There was a well-healed right radical neck dissection scar. The larynx was prominent and the right lobe of the thyroid contained a firm solitary nodule. There was a 1 x 1 cm. firm, rubbery, lymph node palpable just above the superior border of the trapezius just lateral to the posterior cervical triangle on the right. The lungs were clear to percussion and auscultation. The heart was of normal size and no murmurs were detected. There was no significant peripheral lymph node enlargement except as noted above. The abdominal examination was normal. Neurological examination, pelvic, and rectal examinations were normal.

Laboratory data: Hemoglobin 13.4 gms.%, white count, 6,850/mm^3 with 76% neutrophils, 21% lymphocytes, 1% eosinophils and 2% monocytes. Platelets count 162,000/mm^3. An x-ray was reported as showing normal chest and lung fields. A thyroid scan revealed good uptake and good definition of the left lobe of the thyroid but there was decreased uptake in the superior portion of the right lobe of the thyroid and no uptake in the nodular area which had

been previously marked. The lymph node from the right superior cervical triangle was removed and was reported as showing "Hodgkin's Disease of the nodular sclerosing type (variety of granuloma)".

The patient was again seen by Radiation Therapy and given 3800 r to the right supraclavicular and right cervical areas over a period of 34 days with careful observation of the nodule in the right neck. There was no regression of the nodule. The patient was admitted to the hospital on 2-6-66 for exploration of the right neck which was performed the following day.

Clinical Discussion

Dr. Kinloch Nelson: The patient is a 25 year old woman whose history refers to the time she was seen in November 1965. Her chief complaint was an asymptomatic "lump" in the region of the larynx. This should be described with more specific information as to location, size, shape and so forth. This, many times, would be most valuable, here it doesn't really matter. This lump had been present for three months and had apparently been getting larger.

We find that in 1959, at age 19, she had a lump in her neck that was evidently suspected of being a thyroid carcinoma. However, this was found to be a lymph node attached to the thyroid. The tissue diagnosis was Hodgkin's Disease. This diagnosis runs throughout her subsequent history and I think we can be sure that the patient has, and has had, Hodgkin's Disease. In this disease a principal concern is the attempt to make a prognosis from the findings at the time of the original diagnosis. Some think this can be based on the histologic appearance, the location of the lesions, and the presence or absence of systemic symptoms.

Such efforts first came to my attention through the works of Jackson and Parker[1] in 1947. They divided the disease into three stages. The first was *paragranuloma* which was thought to be a relatively benign condition. In this group there were probably patients who did not really have Hodgkin's Disease; such a situation often arises in examples of any disease in which the course and prognosis turns out to have been unusually good. The second category, *granuloma,* is now staged in different ways by different authors. The outlook was not so good and this was related to the area the lesions were in, and how many body areas were involved. In the third stage, *Hodgkin's sarcoma,* the outlook was particularly bad. It turns out that such prognostication is really not very accurate; however, as study and classification[2] of individual cases are better done, the outlook may be improved.

In any case, I judge from the original findings and subsequent course that our patient had one of the least aggressive types of Hodgkin's Disease.

She was treated with radiation and remained well until 1962 when she had lymph nodes in the neck again and this time had a radical neck dissection. The tissue was specifically called Hodgkin's granuloma. We wonder why she had the radical neck dissection. This illustrates the fact that there is little firm opinion as to how to treat individual cases. She had gotten along well for three years while her disease was localized; perhaps there was some hope that the process could be totally removed. As a general principle in Hodgkin's Disease, this is a hope rather than a reality.

In 1962 she had some unbiopsied node enlargement in the left axilla, was treated with x-ray, and responded well. In 1963 she had mediastinal involvement which cleared after x-ray. Could we see the films, Dr. Gildersleeve?

Dr. Gerald Gildersleeve: (Associate Professor, Department of Radiology)

There are 14 sets of x-rays since 1962. In the period referred to in January and February 1963, the right hilus of the patient was enlarged briefly and then returned to normal again following radiation therapy (fig. 1). There had been no pathologic

changes other than that through February 1967. There is no intravenous pyelogram study, just 14 chest x-rays!

Dr. Nelson: The physical examination at the November 1965 admission showed what may have been a slight elevation in temperature; her pulse was a little fast; her weight was ample. She had a scar on her neck, a prominent larynx and a solitary nodule in the region of the right lobe of the thyroid.

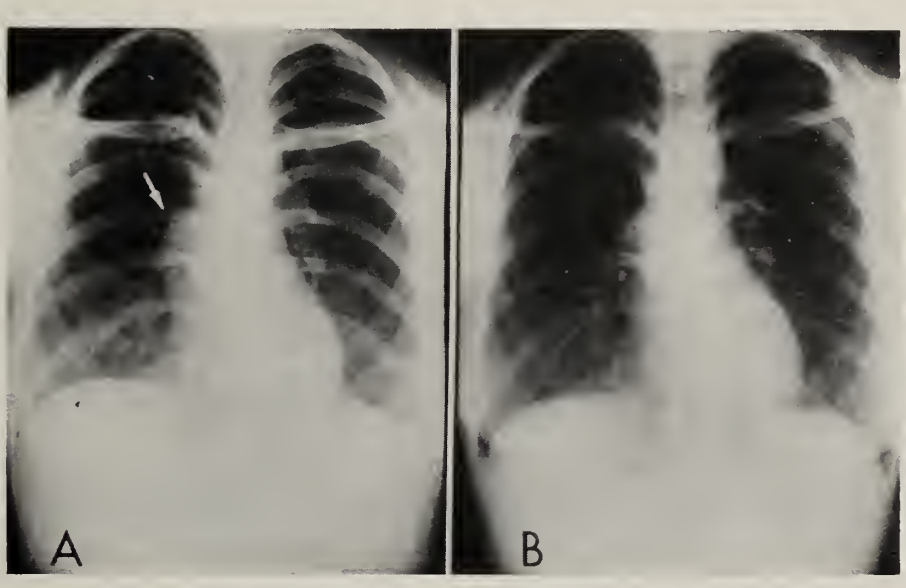


Fig. 1. (a) Hilar adenopathy (arrow) appeared in February 1963 and received radiation therapy. The chest films have since been normal (b).

I would like to caution you about taking these kinds of data too seriously. It has always amazed me that from examining the thyroid, doctors arrive at conclusions which lead them to therapeutic procedures which are obviously ridiculous. I mean the conclusions are; the procedures may be also.

For example, Mortensen[3] and associates reported on 1000 thyroids from consecutive post-mortem examination. After eliminating those patients whose records suggested any kind of thyroid disturbance, he was left with 821 patients whose thyroids were examined both by palpating the removed gland and by sectioning it to see its nodularity or lack thereof. He found that in about one of eight patients with clinically normal thyroids, the gland contained a solitary nodular lesion. These were removed glands which felt non-nodular. About three of eight contained multiple nodules; in only half of the patients was the thyroid actually non-nodular. So, *your* thyroid is full of nodules! That is, 50% of you! Thus, palpation of the thyroid gland even after removal at autopsy is not reliably indicative of the presence or absence of nodules, or how many may be present. 649 of Mortensen's 821 patients had non-nodular thyroids on routine clinical examination, but only 415 of these were non-nodular on section. So, when you examine the thyroid or some region you think is the thyroid and think you feel a nodule, you probably do, and the chance that there are more nodules is very great. Obviously it is ridiculous to base your treatment on your finger-tip—that the thyroid contains a single nodule. And yet this is frequently done.

Our lady had a mass in her neck in the posterior triangle also. In the laboratory nothing spectacular was developed. The fact that she had a platelet count struck me as odd until I read[3] further on what should be done nowadays about examining patients with Hodgkin's Disease; then it struck me as odd that she hadn't had more tests.

There was no B. M. R. Let me take this

opportunity to say "farewell" to this test which has been discontinued at M.C.V. For many years it has been a friend of the doctor, and a great money-maker. Its interpretation was always difficult. Specifically, I recall many thin, "nervous" young women with B. M. R.'s of -20, -18. I never knew what to make of this. In assessing thyroid function we are now relying on the determination of blood levels of free thyroxin, the T_3 resin test, measurements of the protein-bound-iodine (PBI) and the RAI^{131} uptake.



Fig. 2. The scan of the thyroid area following administration of radioactive iodine (I^{131}) was interpreted as showing normal uptake in the left lobe, and decreased uptake in the upper portion of the right lobe. There is no significant uptake in the encircled zone where the mass was palpated.

Dr. Alton Sharpe: (Associate Professor, Departments of Medicine, and Radiology)

We did a scan on this patient after oral administration of radioactive iodine. We interpreted this (fig. 2) to show fairly homogenous uptake in the left lobe, decreased uptake in the superior portion of the right lobe and no uptake in the area outlined. Our interpretation is that there is a mass in the neck which isn't concentrating radioactive iodine. This may or may not be thyroid tissue; it may be thyroid tissue which has undergone malignant change. One cannot make a pathological or histological diagnosis from this picture. One can only say that the region under consideration is not concentrating radioactive iodine.

Dr. Nelson: So, she had a thyroid scan. Uptake was diminished in the upper region on the right, and absent in the nodular area. Our Dr. J. M. Harrison has made an exhaustive study of this procedure in our hospitals over the past ten years; he indicated that findings of this sort are strongly suggestive of neoplastic change and on the contrary, if the so-called "nodular" area takes up I^{131} well, the chances of malignant change are much less. This scan indicates that there are non-palpable nodules above the palpable one; this simply bears out what was said above.

Since this is the point at which the work-up was apparently discontinued in this case, let us review what is now regarded by some as the proper work-up of a patient with lymphoma. First, there should be histologic proof of the disease. In the history particular attention should be paid to whether or not there is evidence of systemic involvement such as fever, night sweats, weight loss, loss of appetite, itching, weakness, cough and bone pain. A physical examination should be done. Laboratory studies should include a hemogram, platelet, and reticulocyte count, stool for blood, urinalysis, bone marrow examination, liver and kidney function tests, and uric acid. Why would an uric acid be ordered in a patient who was thought to have Hodgkin's Disease, Dr. Regelson?

Dr. William Regelson: (Professor and Chairman, Division of Medical Oncology)

The nuclear breakdown products following chemotherapy for malignant tumors may precipitate gout in the potentially susceptible patient. One orders an uric acid to

know where he stands, a base line, in an attempt to anticipate the possibility of precipitating gout. You can avoid this type of gout by using something like Allopurinol which blocks the formation of uric acid.

Dr. Nelson: X-ray examination in the form of lymphangiography, inferior vena cavography, and intravenous pyelography[4] are widely recommended to determine whether or not there is involvement of other lymph node groups. This changes the classification of the patient's staging, so to speak, and thus changes the prognosis and perhaps the therapy. Other procedures of some value in a complete work-up for lymphoma or possible lymphoma are serum protein electrophoresis, skin testing, tests to establish abnormal antibody production, Coombs test, seriological tests for syphilis, rheumatoid factor, lupus erythematous preparation, and protein-bound-iodine.

X-ray treatment was given over the neck which had no effect on this nodule which was in or near the thyroid. We anticipate almost certain response if this were a Hodgkin's lesion or other lymphoid mass.

We conclude from all this, then, that the patient had Hodgkin's Disease which had been radioresponsive and from which she was getting along very well. Now she has an additional problem in the region of the thyroid. If one presumes this to be a single nodule, which I don't, and if one presumes that "cold" nodules are more likely to be malignant than "hot" nodules, and this was a "cold" nodule, then one must wonder if this was a carcinoma of the thyroid. I'll say this lady had Hodgkin's Disease and a lesion in her thyroid which we suspect is a second malignancy.

Dr. Nelson's Diagnosis:

1. *Hodgkin's Disease*
2. *Carcinoma of the thyroid.*

Pathological Discussion

Dr. Fairfield Goodale: In the past several years Dr. Nelson and I have struggled with some weird plagues and pestulentia. On the first two occasions, we were dealing with peculiar vascular disorders which didn't fit into any category that I knew of, but in each instance Dr. Nelson made the correct diagnosis. Therefore, one was *Nelson's Disease,* the other *Nelson's Syndrome.*[5] These are indeed rare afflictions and I have not seen a case of either one before or in the intervening years. But this really should not disturb us very much because sometimes years and years go by while diagnoses lie fallow, until the time when they become household names. We have a very good example in one of the diseases we are discussing today—Hodgkin's Disease.

Hodgkin's Disease, as we know it today, was actually first described 200 years before Hodgkin was born, by a man named Malpighi. Hodgkin, in the 1830's described seven cases of diffuse lymph node and splenic enlargement and then for thirty years his report lay in the archives of Guy's Hospital until some man, bent on publicizing the hospital, published it under the name of "Hodgkin's Disease".[6] It is interesting that when recently the tissues from these seven cases were re-examined histologically, five of them turned out to be tuberculosis or some form of granulomatous disease.

Last year we were spared the necessity of thinking up a new term for the case Dr. Nelson discussed because the lesion had been first described in London 300 years ago by a vascular physiologist, William Harvey. This year, also, the diagnosis is not as difficult as is conjecture on the pathogenesis and the etiology of the condition we are dealing with.

In 1959 the patient's first biopsy was made at another hospital; her Hodgkin's lymphoma then had exactly the same appearance as in our present biopsy. This is a relatively calm histologic appearance for Hodgkin's Disease. Almost all the cells are adult lymphocytes. There are some histiocytes; Reed-Sternberg cells are relatively few and far between (fig. 3).

I agree with Dr. Nelson that the Jackson-Parker classification of lymphoma leaves a great deal to be desired when it comes to anticipating the prognosis. Lukes[7] has recently described what may well be a better classification. In essence, he divides Hodgkin's Disease into the quiescent and progressive forms. In the quiescent form survival time is really quite favorable. In these patients the histologic material shows considerable lymphocytic and histiocytic proliferation. There are relatively few Reed-Sternberg cells and if there is fibrosis, it is focal. With the progressive form survival time is relatively short and in these patients there is lymphocytic depletion, more Reed-Sternberg cells, and the fibrosis tends to be diffuse. Of course, there are stages between these.



Fig. 3. Hodgkin's Disease as seen in the original biopsy. A uniform infiltration of relatively mature lymphocytes obscures the normal lymph node architecture. The large Reed-Sternberg cells are sparse. (hematoxylin and eosin; magnification from 45X)

So today's patient developed, at age 19, Hodgkin's Disease of a type which fits Lukes' quiescent form. She received irradiation to the neck and upper chest at that time, at age 22, at age 23, and again just before her last admission. Now she has developed a second malignancy: carcinoma of the thyroid. This was a typical papillary lesion with psommoma bodies (fig. 4), the most common type of thyroid carcinoma. With the mixed follicular and papillary carcinoma, which has the same behavior, it constitutes 70-80 per cent of cases of thyroid cancer. This group has the best prognosis of the various thyroid malignancies.

There are many published series of patients in whom thyroid carcinoma has developed following irradiation for one purpose or another.[8,9,10] The irradiation has been given to shrink tonsils and adenoids or to shrink the thymus; it has been given to treat scrofula and acne, and to irradiate cervical lymph nodes in lymphomas such as Hodgkin's Disease. Of patients in whom the histologic diagnosis of thyroid carcinoma was made before they were 25 years old, almost half gave a history of previous irradiation: of patients under 17 years of age, almost all had previous neck or upper tho-

rax "therapeutic" irradiation. If we look at it the other way around and follow children who have had such irradiation to see the frequency of development of thyroid carcinoma, we find that, depending on whose series you read, there is an increased incidence of eight to 40 times that of control patients. Obviously then, radiation, particularly in children, to the region of the thyroid would seem a particularly dangerous procedure. I would like to ask Dr. King if he would comment on this aspect of the case. I would also like to ask him if he would give us his criteria for believing that a carcinoma developing after radiation is really related to that radiation.

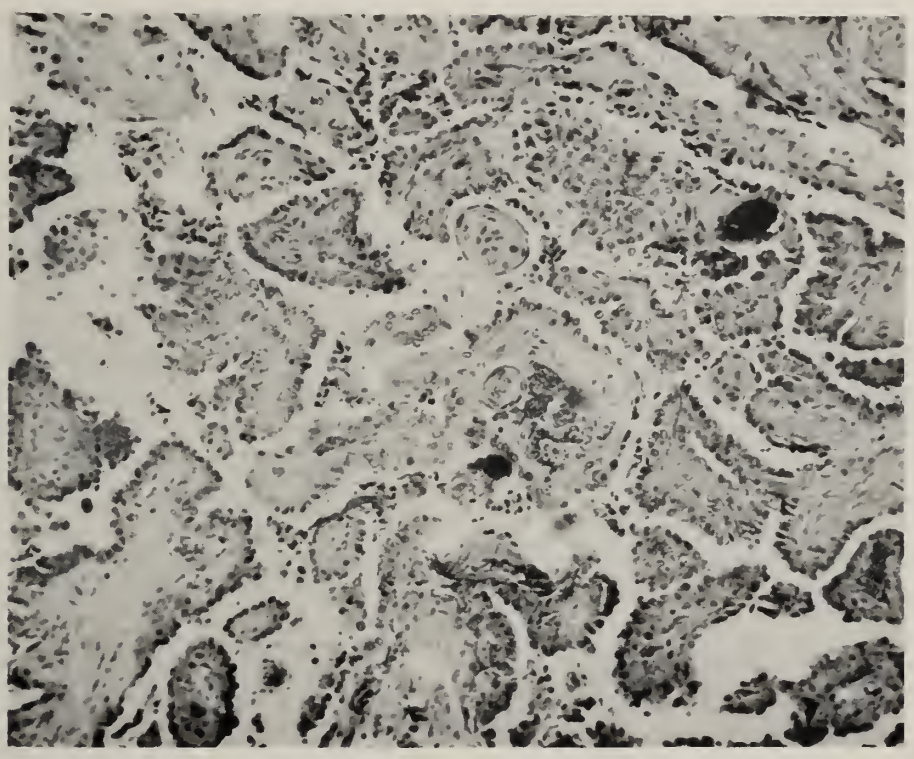


Fig. 4. Papillary carcinoma of the thyroid. The dense, calcific masses are "psommoma bodies". (hematoxylin and eosin; magnification from 140 X)

Dr. E. Richard King: (Professor and Chairman, Department of Radiology)

I would like to discuss very briefly the role of radiation in the treatment of Hodgkin's Disease before we go into Dr. Goodale's questions. During the past several years, the approach of Hodgkin's Disease by radiation therapy has undergone a revolution. At the present time, in the early stages of Hodgkin's Disease or fairly localized Hodgkin's without systemic effects, we treat the contiguous node-bearing areas as well as the primary site. For example, if the lesion is above the diaphragm, we usually treat the mediastinum, both axillae, supraclavicular areas, and cervical areas. In our institution, we spare the thyroid gland. If the lesion is in the mediastinum we usually move the treatment field down to include the periaortic nodes and occasionally the pelvic node areas. The dose given in the larger field of treatment like this is around 4,000 to 4,500 rad. I might say that this large treatment field is possible only by high energy x-ray such as the two million kilovolt Maxitron of the Cobalt 60 unit we have here and have been using since 1961.

Let me outline roughly the treatment that was used on this patient. In area *1* (fig. 5), the first treatment she received was by low-energy x-ray in 1959. This was all that was available at MCV at that time

so it was impossible to treat the contiguous node-bearing areas. *II* is the treatment field she received in 1962 which included not only the axillary areas where the node was, but also the supraclavicular and cervical regions. *III* is the mediastinal field in 1963. *IV* is a rather large treatment field in which we actually included the node in the thyroid gland. I must say I did this against my better judgment. I followed this patient for some

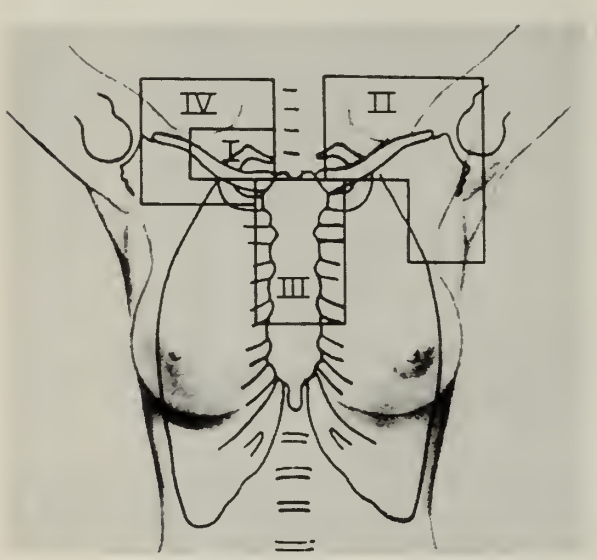


Fig. 5. Outline of irradiation treatment fields. See text (Dr. King).

time during the summer and fall and the lump in the lower pole of the right lower pole of the thyroid was very evident. It was hard, well outlined, and felt like a malignancy to me. She had had a radical neck on that side and did have a neck deformity as often occurs after that procedure. At least one other examiner thought that the lump might be the end of the sternocleidomastoid muscle. Nevertheless we did treat her and the lump did not respond. In fact, it grew larger during the course of radiation therapy.

There is no doubt but what infants and children who have had previous radiation, even in small doses, have a much higher incidence of thyroid carcinoma than in non-irradiated "control" groups. This tends to implicate radiation as a carcinogenic agent in cancer of the thyroid. But, as far as I know, this has only been reported in young people. By young people, I mean people usually under the age of 10. Also, this usually takes a few years to develop. I don't believe in the literature that there have been cases reported of cancer caused by radiation with a latent period of less than five years. In this instance, it was six or seven years.

We did not directly irradiate the thyroid in 1959 and really the beams were very well outlined and clearly defined. One does not ordinarily get enough scatter to do any harm, unless there is something such as an abscopal effect. I can't say that radiation wasn't involved here, but I rather doubt its importance in the etiology of the thyroid carcinoma.

Dr. Goodale: Dr. Regelson, do you consider Hodgkin's Disease as a malignancy or as an infectious process? I happened to look up, last night, in the unabridged Random House Dictionary of the English Language, the definition of Hodgkin's Disease. It reads: "A disease characterized by progressive, chronic inflammation and enlargement of lymph nodes—and spleen". Should I continue to use this dictionary? Perhaps as a doorstop?

Dr. Regelson: Well, your talking about Hodgkin's Disease as being an infectious disease relates to the thyroid because Dr. Jacob Fürth's first experience with thyroid stimulating hormone as an inducer of thyroid cancer set back the recognition of the viral etiology of cancer by many, many years. Fürth originally came into the field with a great deal of enthusiasm for the virus concept of cancer. He found, however, that excessive stimulation of the thyroid by TSH results in a very high incidence of thyroid cancer in specific strains of mouse and rat. His enthusiasm for these results diminished his interest in accepting the viral etiology theory.

Getting back to lymphoma as "infectious disease", certainly in mice there are as many as 60 different virus strains that can induce leukemias or lymphomas. I'm working with a Friend-virus which produces a reticulum

cell sarcoma in a mouse within four days of interperitoneal innoculation of the virus. We have now studies in leukemia that show epidemiologically a pairing phenomenon of associated cases, "little clusters" of leukemia. In cattle we have the bovine lymphomatosis which has many characteristics of human lymphomas, including Hodgkin's. There are virus particles found in the milk of cows that carry this particular strain of lymphoma. Thus, we are getting back to lymphomas being of viral etiology, but how that shapes up in relation to calling it an "infectious" disease is uncertain at this point.

I'm willing to bet that Hodgkin's Disease is a contagious disease and that we all have been exposed to its viruses. The key to the difference between this girl who at age 19 gets the disease and you and I who haven't had it, or won't get it, or haven't gotten it yet, is this factor of host resistance. For example, in my mouse system, I can turn on or off the resistance of a mouse to the Friend-leukemia by pre-treatment with a variety of agents. Pertussis vaccine increases the susceptibility to leukemia; synthetic polymers, and some other compounds which stimulate the reticuloendothelial system, increases resistance.

Dr. Goodale: Dr. Regelson, can radiotherapy cure Hodgkin's Disease?

Dr. Regelson: I think it is malpractice if one doesn't radiate contiguous node-bearing areas in Stage *I* and Stage *II* of Hodgkin's Disease. We are neglecting our Hodgkin's cases by not enough doctors giving this pattern of radiotherapy. I speak about this with conviction because I was trained under Lloyd Craver at Memorial Center. At that time he taught that Stage II Hodgkin's Disease is not curable. We gave 3,000 rads to the local area just to shrink nodes for cosmetic or mechanical purposes and we accepted the "fact" that we couldn't "cure" these patients. Now, the evidence is quite clear that the pattern of five years and 10 years survival, particularly for Stage *I* and *II* is good and that the Radiotherapist can, I think, cure Hodgkin's Disease.

Dr. Goodale: Dr. Hudson, experimental induction of cancer has been an interest of yours. Have you any comment concerning the possibility of this thyroid carcinoma being related to the previous radiotherapy?

Dr. Page Hudson: (Associate Professor, Department of Surgical Pathology)

I believe that the thyroid carcinoma was induced by the radiation received years before. Dr. King's authority and knowledge are greatly respected with good reason. However, I have followed this case carefully beginning with frozen section examination at surgery.

The number of reports of thyroid carcinoma following irradiation in children and young adults continues to grow. Hagler[8] recently reviewed the subject and reported 18 new cases. He pointed out that 80% of 277 cases of thyroid cancer in this age group had a distinct history of irradiation in the appropriate area. Another approach, as you noted, Dr. Goodale, was to determine what proportion of infants and children given radiotherapy about the neck and upper chest developed thyroid carcinoma. Hagler's review of other studies indicated an occurrence varying from one in 150 to one in 867. This was a much higher rate than in untreated siblings or the general population.

That thyroid cancer can occur in adults who, as adults, had radiotherapy in the thyroid region has been reported by Goolden[11] and others. The report of Lindsay and Chaikoff[12] is another superb review.

Evidence in our case today that irradiation may have induced the tumor is provided by examination of the non-tumor bearing portion of the gland. Dr. King considered that the patient's thyroid was shielded. However, there is much microscopic atrophy and scarring and even mild atypism in areas of the gland distant from those bearing the carcinoma as well as those adjacent to the tumor. These changes

strongly suggest radiation effect rather than changes produced in the gland by neoplasm or some other process. The changes were similar to those seen in the thyroids of irradiated rate in whom thyroid carcinoma developed.

I believe the irradiation given as therapy for Hodgkin's Disease very likely induced the example of thyroid carcinoma under discussion today.

Dr. Goodale: The sequence of events in this patient starts with Hodgkin's Disease in cervical lymph nodes treated with radiation therapy, and goes on to the development of papillary carcinoma of the thyroid from which there were no lymph node metastases at the time of operation. Both of the malignant processes in this girl are apparently indolent and slowly progressive. The question that now is of considerable concern is which of these malignancies, if either, is going to catch up with her first.

Pathological Diagnosis:

1. *Hodgkin's Disease.*
2. *Papillary carcinoma of the thyroid, probably induced by irradiation therapy for Hodgkin's Disease.*

Please address reprint requests to Dr. Page Hudson, P.O. Box 871, Medical College of Virginia.

Illustrations were prepared thru the facilities of the Department of Visual Education, Medical College of Virginia, Mr. Melvin C. Shaffer, Director.

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Mental Health . . . .

HIRAM W. DAVIS, M.D.

Geriatric Patients in Virginia's State Mental Hospitals

As of the end of June, 1967, 30.8% of the resident population in the four major State mental hospitals was 65 years of age or older. Contrary to trends reported by many other States, this percentage is slowly rising in Virginia. This represents in round figures some 3,500 persons. While many of these are in need of psychiatric care, it is undoubtedly true that a relatively high percentage are persons with minor psychiatric overtones who could, if proper alternative facilities outside the hospital were available, be placed in non-psychiatric institutions or kept with their families. It is considered that many are placed in the Mental Hospitals of Virginia because of the good level of care provided and because it is less costly to the individual or his family.

Basically, the resident mental patients over 65 may be broadly classified into three categories:

1. Those physically in good condition, ambulatory, and able to participate fully in treatment and activities to the same extent as the majority of other patients.
2. Those with some physical limitations, debilitated to some extent, limited in their activities but able to be ambulatory through the use of wheel chairs or crutches.
3. Those chronically ill who are bedfast and require almost constant nursing care and general medical attention.

It is these latter patients who place a disproportionate burden upon the staffs of the hospitals because they must be moved, bathed, clothed and, in many cases, fed by the nursing personnel. Because these patients require a higher staffing ratio, they impose an economic problem for the hospital due to the higher per diem cost of their treatment. If some solution could be found for the removal from the hospitals of these elderly, senile and, in many cases, only mildly disturbed patients and at the same time the same staffing levels maintained at our hospitals, the overall level of care and treatment of the severely mentally ill could be increased considerably.

In effect, elderly patients over the age of 65 have become a responsibility of the Department of Mental Hygiene and Hospitals through default because sufficient proper facilities at the same cost are not available elsewhere.

Until two years ago, regulations regarding private nursing homes licensed by the Department of Health precluded the admission of patients with a psychiatric diagnosis. This has been changed and such patients may now be admitted with certification that they do not constitute a danger to themselves or others. It is possible that a portion of the hospital load could be shifted to nursing homes. However, the cost of nursing home care is relatively expensive in itself and normally does not include physicians' fees, necessary special medical attention or drugs. Few of these homes offer charity privileges. Nevertheless, the number of beds in nursing homes so licensed has increased since 1957 from 3,947 to 6,972. A continued increase, coupled with the proposed expansion of long-term care units in General Hospitals could well relieve mental health programs with respect to the elderly.

Other resources of more limited nature are the homes for the aged licensed by the Department of Welfare. At present there

DAVIS, HIRAM W., M.D., *Commissioner, Department of Mental Hygiene and Hospitals.*

are approximately 4,500 beds available through proprietary, church-sponsored and local government operated institutions. The majority do not provide intensive nursing care and are basically limited to ambulatory or light nursing care patients. Welfare Departments share in the cost or provide full compensation to many patients in such homes. Some of these specialize in the care of former mental patients with a high degree of success.

Despite the availability of the above, one must realize that for Virginia with an overall population of 4.5 million people and a growing percentage of the elderly, provision of only 12,000 such beds for all geriatric patients or residents of whatever infirmity leaves the Commonwealth woefully short of local facilities to alleviate appreciably the situation at our hospitals.

To meet the problem which currently faces it the Department has made a two-pronged attack.

First, there has been a concerted drive to increase the staffs of the hospitals both quantitatively and qualitatively. This is a prime objective, not only for the benefit of the geriatric cases, but for all patients. A series of salary increases, inservice training programs, use of psychiatric practical nurses, and recruitment of personnel of all disciplines have resulted in a creditable increase in employee-patient ratios. While still lower than would be desired, conditions in this regard have improved over the past ten years.

The Department's efforts toward accreditation of all hospitals will also be a factor which will improve the care of all patients.

Second, the Capital Outlay Program has been designed to relieve overcrowded conditions in all hospitals and to provide modernized facilities. In the case of geriatrics construction, this has resulted and will result in buildings designed specifically for elderly patients. An example of this type of construction is the new Hancock Geriatrics Complex at Eastern State Hospital incorporating 550 beds which will make it possible to move all remaining elderly cases from the outmoded and overcrowded buildings remaining at the Williamsburg site. Other State Hospitals have similar construction and renovation programs.

On July 1, 1967, the Department took over administration of the former Tuberculosis Hospital, Piedmont Sanatorium, at Burkeville, which will add 300 beds to the system. It is planned that this new State Hospital will be utilized primarily for geriatrics cases transferred from other State Hospitals with a view to moving them closer to their home.

Recognizing its responsibility not only for the care and treatment of elderly patients placed in its care, but also for assisting in reaching solutions to the overall problem, the Department has requested funds for the establishment of the position of Geriatrics Program Director at the Central Office level.

If the General Assembly continues its past support in terms of capital outlay and in maintenance and operation funds, the lot of all patients in our hospitals will continue to improve and that of the chronic debilitated geriatric patient will certainly be bettered in all respects.

The Department of Mental Hygiene and Hospitals has been assigned and has assumed a major role in the geriatrics field in Virginia. It recognizes, however, that this responsibility is being shared by many other agencies and groups in the Commonwealth and that through the Governor's Commission on the Aging impressive efforts are being made locally and at the State level to better the conditions of all of its senior citizens. Considerably more can be done at the local level. Programs can and must be developed to include adequate recreational and social facilities for the elderly and job opportunities for the retired. In the Mental Health field, increased utilization of local mental hygiene clinics and, in the future, Comprehensive Community Mental Health Services

and Centers should serve to reduce the numbers who are sent to our State Hospitals.

The State Hospital Board, the Department of Mental Hygiene and Hospitals, and the Commissioner, recognizing the tremendous services and responsibilities involving the sick and disabled geriatric citizens of our State, have recommended that a Special Commission for Geriatric Patients be established by the 1968 General Assembly to study and make a report with recommendations to the Governor and the 1970 General Assembly. The Commission Report should include all phases of inpatient and outpatient services, boarding homes, nursing homes, day care facilities and any other type of facility which would be available to provide services for the older residents of Virginia.

Clinical Center Study of Malignant Lymphoma

The cooperation of physicians is requested in a study of lymphosarcoma and reticulum cell sarcoma in children and young adults. This study is being conducted by the Medicine Branch of the National Cancer Institute at the Clinical Center, National Institutes of Health, Bethesda, Maryland.

Slides of pathologic material must be submitted for review before patients can be accepted.

Of particular interest are those patients with clinical and histologic features similar to the malignant lymphoma of African children (Burkitt tumor). These patients generally present with jaw, ovarian, or abdominal masses. The purpose of this study is to conduct immunologic, virologic, pathologic, and chemotherapeutic studies.

Suitable patients will be admitted to the Clinical Center in Bethesda, Maryland, or to one of the participating medical centers. Physicians who wish to have their patients considered for the study may write or telephone: Paul P. Carbone, M.D., The Clinical Center, National Institutes of Health, Building 10-Room 12-N-226, Bethesda, Maryland 20014. Telephone: 656-4000, Ext. 64251 (Area code 301)

Diagnostic Laboratory Medicine....

J. B. ELLIS, M.D.

Trichinosis

Trichinosis is produced by *Trichinella spiralis,* and aphasmid nematode parasite of man. Man consumes raw or rare pork infested with cysts. Digestion results in encysting in the duodenum of larvae, which invade duodenal and jejunal mucosa and develop into adult worms within five to seven days. After fertilization, female worms burrow into the intestinal wall and deposit larvae over a period of approximately four to sixteen weeks. The larvae enter lymphatics or venules and thus are distributed, between the ninth and twenty-third days, throughout the body and upon reaching striated muscle, encyst. Encysted larvae may remain viable for many years, although calcification usually occurs within six to nine months.

Pathology and symptomatology are variable but in the typical case progress through three stages. The first stage, that of invasion of intestinal mucosa, is associated with nausea, emesis and diarrhea. The second stage, migration, is manifested by fever, eosinophilia (usually 15-20%), muscular pains, edema, lymphadenopathy, occasionally myocarditis, meningitis or encephalitis. The third stage, encystation, is the most critical and is associated with cachexia, generalized toxemia, edema and dehydration. Death may occur, the individual being in a state of shock with cyanosis.

In the majority of cases, however, symptoms are absent or so mild as to lead to no specific diagnosis; Link in 1952 estimated that of 350,000 new cases yearly in the U.S.A., only 16,000 had symptoms. Recent investigations indicate decreasing incidence and severity of trichinosis in the United States. In 1930, the incidence of trichinosis noted at autopsy was between 15% and 25%. In the 1960's, the reported incidence at autopsy was 5%. In the past the mortality in cases having neurological or cardiac involvement was 50%; at present it is 8%. Improvement is due to better feeding practices by pork raisers and improved methods of preparation of pork products.

Only rarely are the adult worms recovered from feces; or the larvae from blood, spinal fluid, or other body fluids. The diagnosis is confirmed by muscle biopsy, positive skin test, or positive serological tests.

The usual muscles for biopsy are deltoid, biceps, or gastrocnemius which may demonstrate either unencysted or encysted larvae.

The Bachman intradermal skin test uses antigen in 1:5,000 to 10,000 dilution and is reported to be highly specific. A positive test result consists of the immediate appearance of a wheal (approximately 5 cm. diameter) at the injection site. Reading is made before 15-20 min., since the reaction fades. This immediate-type skin reaction does not become positive, however, until the individual is well into the stage of encystation (approximately three weeks). Earlier in the course there may be a delayed-type reaction which consists of development of 10 to 15 millimeter areas of induration about twelve hours following injection.

Positive serological tests are generally considered to give the most reliable evidence of Trichinella infestation: complement-fixation, agglutination, precipitation, fluorescent antibody, and flocculation tests are done. The Bentonite slide flocculation test, as developed by Bozicevich, is considered specific and highly satisfactory. For this procedure stock antigen is prepared by absorbing Trichinella extract and a basic dye, Thionin Blue O, with a Bentonite suspen-

sion. This is stored at 4° C and is stable for several months. Saline dilutions of test serum plus antigen are rotated for 15 minutes on wax ringed slides and flocculation reactions then are read. Two controls are used, negative serum and saline. Reactions are read from 0 to 4+. A 3+ or 4+ reaction is considered positive and 2+ or less is considered negative.

Summary

A discussion of the life history of the organism *Trichinella spiralis* which is responsible for trichinosis is given. The pathology and symptomatology accompanying the disease, together with its diagnosis and prophylaxis, is presented.

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Division of Clinical Pathology
Medical College of Virginia
Richmond, Virginia

AMA Adopts Expanded Statement of Purpose

Broad initiative in advancing health care for everyone is detailed in an expanded statement of purpose and responsibilities of the American Medical Association adopted by its House of Delegates. The 11-point document greatly enlarges upon the previous statement of AMA purposes. The statement declares that it is the responsibility of the AMA, as the representative of the American medical profession, to continue to foster the advancement of medical science and the health of the American people and to discharge that responsibility through the following means:

1. By encouraging the advancement of medical knowledge, skills, techniques and drugs; and by maintaining the highest standards of practice and health care.

2. By creating incentives to attract increasing numbers of capable people into medicine and the other health-care professions.

3. By advancing and expanding the education of physicians and other groups in the health-care field.

4. By motivating skilled physicians who have the art of teaching to apply themselves to developing new generations of excellent practitioners.

5. By fostering programs that will encourage medical and health personnel to serve voluntarily in the areas of need for medical care.

6. By developing techniques and practices that will moderate the costs of good medical and health care.

7. By seeking out and fostering means of making all health care facilities—physicians' offices, hospitals, laboratories, clinics and others—as efficient and economical as good medical practice and attention to human values will permit.

8. By combing the utilization of the latest knowledge for prevention and treatment with the vital healing force of the physician's personal knowledge of and devotion to his patient.

9. By maintaining the impetus of dedicated men and women in providing excellent health care by preserving the incentives and effectiveness of unshackled medical practice.

10. By maintaining the highest level of ethics and professional standards among all members of the medical profession.

11. By providing leadership and guidance to the medical profession of the world in meeting the health needs of changing populations.

Public Health . . .

MACK I. SHANHOLTZ, M.D.
State Health Commissioner of Virginia

Heart Disease and the Sodium Content of Drinking Water

Sodium is a positively charged ion with an atomic weight of 32. Because of its ability to combine with many substances, it is present almost everywhere. Man thinks of sodium in terms of salt or sodium chloride, which is 40 per cent sodium. This compound is in some of the foods he eats, such as ham or bacon; in the water in which he swims; and in the additions to his diet, such as salt tablets and table salt.

But he may also drink water treated by chemical softeners which introduce comparatively large amounts of sodium without adding a salty taste. Sodium in the form of salt can be useful to man, but at the same time might be life-threatening to the congestive heart failure patient. Thus, the knowledge of the sodium content in drinking water is important to the management of these patients.

Congestive heart failure is a complication of every form of heart disease; and 50 per cent of all cardiac patients die of congestive heart failure, which occurs when the damaged heart can no longer pump efficiently due to excessive fluid retention.

The treatment of congestive heart failure is directed toward the problem of unusual retention of sodium by the kidneys. A sodium-restricted diet is prescribed to decrease the amount of ingested sodium, and a diuretic may be given to promote excretion of sodium and water. In addition, digitalis may be given to increase the heart's efficiency. Some or all of these methods of control may be instituted, but in almost every case, some degree of sodium restriction is recommended. In any case, the sodium content of water is of considerable importance.

Water supplied to users comes from many sources—streams above and below ground, wells at different levels, pooled and single source reservoirs. Different types of processing all add or subtract such substances as sodium. The source water is invariably of different composition from the finished water. It has been determined, however, that such treatment as fluoridation can be considered insignificant even for patients on a sodium-restricted diet.

The fact that sodium is present in drinking water is not news, and the fact that it may be present in high concentrations has been known for over twenty years. What is not known is whether or not seasonal variations of the sodium content of drinking water are significant.

The Virginia State Department of Health cooperated with the U. S. Public Health Service in a nation-wide sampling of the sodium content of drinking water. The study lasted for two years—1963-1965—and included 3,000 U. S. cities and towns. Data from the completed study revealed very few communities in Virginia with a high sodium content in their public water supplies.

One community's high sodium content was found in the operation of a softener which regenerated with sodium chloride. During the passage of the town water through the softener, the calcium and magnesium were removed or reduced, and sodium was given up to the water. This source of supply and softening plant have been abandoned, and a new water filtration plant for treating surface supply from the river is


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now in operation. Further study is planned for those other communities in Virginia with an elevated sodium content in their drinking water in hopes of determining the reasons.

Data are available on 44 communities throughout Virginia which were tested during the study period. Any interested physician may obtain information regarding the sodium content of drinking water in his locality from his local health department or by contacting the Virginia State Department of Health, Bureau of Chronic Disease Control, Richmond, Virginia 23219.

MONTHLY REPORT OF BUREAU OF COMMUNICABLE DISEASE CONTROL

	Nov. 1967	Nov. 1966	Jan.-Nov. 1967	Jan.-Nov. 1966
Brucellosis	3	2	38	24
Diphtheria	5	0	5	0
Hepatitis	47	51	683	523
Meningitis (Aseptic)	36	1	59	30
Meningococcal Infections	2	3	41	71
Poliomyelitis	0	0	0	0
Rocky Mt. Spotted Fever	0	0	29	32
Rubella	20	9	682	935
Rubeola	17	33	2220	2236
Streptococcal Sore Throat (including Scarlet Fever)	982	915	12763	11384
Tularemia	0	0	0	2
Typhoid Fever	1	2	5	15
Rabies (in animals)	6	16	187	243
Venereal Diseases				
Syphilis	162	147	1861	1860
Gonorrhea	753	738	8888	7889
Other	0	2	19	29

Woman's Auxiliary...

A Gathering of the Clan atmosphere prevailed at Miami Beach for the meeting of the Southern Medical Association and its Auxiliary, November 13-16. Our Auxiliary was ably represented by Mrs. W. Nash Thompson, Virginia's Councilor; Mrs. Daniel Anderson, our President; and by our President-Elect, Mrs. Robert Keeling. The weather was warm as was the friendly atmosphere. The hospitality room on the Eden Roc mezzanine provided a place to meet and view the state exhibits for Research and Romance of Medicine and Doctors' Day Observance—the two major projects of the Southern Auxiliary. Mrs. Thompson had a fine exhibit and received an Honorable Mention Certificate. Congratulations to you Opal! Hats off to Mrs. Gerszten of the Richmond Auxiliary for her fine exhibit "Deans of the Century" which also capped an Honorable Mention Certificate.

Dr. Southgate Leigh of Norfolk was the featured speaker for the annual Doctors' Day Awards Luncheon. As Chief Surgeon of Seaboard Air Line Railroad (son of Dr. Leigh—honored by our State Leigh-Hodges-Wright Fund), he reminisced on early years of the Auxiliary. Telling of childhood experiences of traveling to medical meetings by pullman—with a governess—he outlined briefly the leadership activities of his family. They were organizers of the Norfolk and Virginia Auxiliary in 1922—and were also instrumental in organizing the National Auxiliary that same year. He spoke of the pride with which his mother's friend had shown the prison uniform she had worn in the cause of Women's Suffrage. "Such has been the leadership shown by Southern Doctors' wives through the years," he praised.

Recalling his mother's service as President of the Woman's Auxiliary to the Southern Medical Association in 1934, he read some of her hand-written notes on Southern Auxiliary stationery which included the following poem:

The Doctor's Wife

Who plans the meals?
Who tends the phone?
Who goes to church alone?
Who worries most?
Who wins the row?
None other than the doctor's frau.

by Dr. Rock Slyster

Dr. Leigh concluded with a verbal salute to the Auxiliary leadership in Virginia and throughout the South—particularly to Mrs. C. Talbot Wilkinson of Wake Forest, North Carolina, the retiring President of The Woman's Auxiliary to the Southern Medical Association.

IN MEMORIUM

Gertrude McIlroy Burger Harris

It is with warm regard and real affection that we remember Mrs. Gertrude McIlroy Burger Harris, who died this November in Norfolk, Virginia.

Mrs. Harris, the widow of Dr. William Lett Harris, served two consecutive years as President of the Woman's Auxiliary to The Medical Society of Virginia 1932-33 and 1933-34. Under her guidance the Woman's Auxiliary to the Alexandria Medical Society was organized.

We pay tribute to a lovely lady of many accomplishments.

To Advise and Consent . . . Dr. Thomas W. Murrell, President of The Medical Society of Virginia, has appointed an outstanding Advisory Council to our Auxiliary. Dr. K. Kenneth Wallace of Norfolk, immediate past president, will serve as Chairman; Dr. F. Ashton Carmines of Newport News, President-elect of the Medical Society; Dr. Malcolm Harris of West Point, Dr. K. K. Wallace, Jr., of Virginia Beach and Dr. Carl E. Stark of Wytheville have also agreed to serve. The Auxiliary is most fortunate to have doctors of such professional statute to guide us. Any committee chairman can be certain that this Council is available to her questions and will give all problems, presented to them, sincere and objective consideration. Another "Treat —Not A Treatment" for our Auxiliary year!—Mrs. Daniel Anderson, President.

Helping Hands to Belroi are extended by a new Auxiliary committee chairmanned by Mrs. Raymond Brown of Gloucester. The Walter Reed Birthplace (Belroi) Committee is most enthusiastic for the long-range project. Mrs. F. Ashton Carmines of Newport News, Mrs. Joseph Chinn of Tappahannock, Mrs. F. H. McGovern of Danville, Mrs. Paul Pearson of Warsaw and Mrs. Nash Thompson of Stuart are also working on this committee. They plan to catalogue the furnishings needed and depend on each Auxiliary member around the State to help locate them. Donations from individuals as well as from antique dealers and auction houses will be greatly appreciated and will be appropriately noted on the article. Articles lent on a long-term loan basis will also be appreciated. The Medical Society of Virginia owns this old home which was the birthplace of Walter Reed, his family having sought refuge there after their home, the manse, was burned. The Medical Society Committee, chairmanned by Dr. Raymond Brown, has asked our assistance which affords us a real opportunity in preserving another landmark of our great medical and historic heritage in the Commonwealth.

Miscellaneous . . . .

Virginia Association of Medical Assistants

The Virginia Association of Medical Assistants helds its 12th annual convention at The America House, Petersburg, November 10-12.

Mrs. Charlotte Davis, retiring President, is to be commended on her smooth efficient handling of this meeting and the manner in which she led the organization during her year as president.

The Host Chapter went "all out" to see that their meeting was "tops" in providing Scientific and Educational material that would help their members render greater service to their employers and their patients.

The social and entertainment aspects were provided by the A. & J. Hospital Supply Company of Richmond and "The Knight Kords". Usually, I shudder at the thoughts of an after-dinner speaker but Dr. Simon Tussi, Pathologist of Petersburg General Hospital, made an after-dinner speech that was "out of this world" both from a biblical and chronological standpoint.

The officers for 1968 are: President—Doris J. Jennings, Roanoke; President-Elect—Evelyn D. Camper, Norfolk; Vice-President—Jean Pair, Jarrett; Secretary—Grace I. Haworth, Norfolk; Treasurer—Ruth Garst Showalter, Roanoke.

Advisors to the VAMA include: Dr. John S. Thiemeyer, Norfolk; Dr. William M. Eagles, Richmond; Mr. Robert I. Howard, Richmond; Dr. John Wyatt Davis, Jr., Lynchburg; Dr. William B. Pope, Jr., Portsmouth; Dr. John T. Jarrett, Richmond, Liaison, The Medical Society of Virginia.

The attendance in Petersburg was about 100. This was the largest but it represents a small percentage of the ladies who work in our offices.

G.P.'s—The girls in our offices are the greatest asset we have and are our most productive investment so insist on our ladies joining the local, state, and national organization. Pay their dues, allow them time off to attend ALL meetings and, when possible, help defray their expenses at these meetings. It is all deductible and will cut down on your enforced contribution toward paying "All the Way: for the Great Society of L.B.J."

The Public Relation's Program of the Virginia Association of Medical Assistants offers many points of assistance to us during the trying years ahead. This program includes such subjects as: Booth space at medical meetings; Sponsor a coffee hour for delegates at these meetings; Display at Health Fairs or other similar functions; Promote "Medical Assistant Career" programs; Establish a "Medical Assistant" week in the state; Cooperate with the medical society and allied organizations; Participation in civic, welfare and humanitarian activities; Keep the medical profession informed of significant facts and events of the organization; Formally offer to assist the local medical society in its public relations/community service campaigns; Request space in state journals to report chapter activities; Sponsor a program on a subject of general interest, such as telephone techniques, and invite the physicians in the area to send their employees; Meet with the local or state medical association executive director for a "get acquainted" session. Explain the purposes of AAMA, and VAMA and ask his aid and advice on how to "sell" the association to the physicians.

So you see, fellows, they are going all out to help us—let's meet them at least half way.

The meeting of the American Association of Medical Assistants in Los Angeles was one of their largest, 907 registrants out of a membership of about 14,000. Under the able guidance of Miss Margaret Swank,

President; Mrs. Mildred Crawford, President-Elect; and Mrs. Dene R. Murray, Executive Director, and a very capable Board of Directors in addition to an outstanding Advisory Committee with Dr. Glenn E. Millard, Chairman and Dr. Wesley W. Hall, Chairman of the Board of AMA, who is Liaison Officer of AMA, this organization will certainly grow in significance, prestige, and assistance to the medical profession.

There were about 15 VAMA members in Los Angeles. Let's insist upon the ladies in our offices joining the local, state, and national organizations with a goal of 50 Virginia members at the '68 meeting of AAMA in Columbus in '68.

John Wyatt Davis, Jr., M.D.
AAMA Advisor



Dr. John Wyatt Davis, Jr., received Outstanding Service Award at Los Angeles meeting of AAMA.

Editorial . . .

The Physiological Clock

EXTERNAL TIME SIGNALS are noted in the color change of a certain fiddler crab species (Uca) which is in tune with tidal and diurnal rhythms, as well as semi-lunar rhythm. Even the lowly potato may be shown to have increased oxygen consumption (by the slice technique) during the day, particularly the early morning, and to decrease its oxygen consumption during the night. There is also correlation between external barometric-pressure and oxygen consumption, but it is not so definite as the day-night cycle.

There are now many factors pointing toward biochemical and/or biophysical systems within organisms which may ultimately control the circadian rhythms. We are not clear how these systems work but the hypothesis now extant is based on the fact that the observed rhythms are independently maintained. To complicate things more, there is the obvious fact that there is significant species variation in their diurnal periodic rhythms.

Terrestrial magnetism is shown to fluctuate rhythmically with solar and lunar periods. The paths of certain mud snails will show a linear directional movement varying with the time of day and the lunar cycle. When this magnetic field is changed by artificially increasing an oblique field to that of the earth's, the snails were found to adjust to one field during the day and the other at night. This led investigators to conclude that organisms are at all times under the influence of environmental variables.

The effects of cosmic radiation on elemental life forms have also been investigated and screening with lead shields does not seem to stop the changes.

In a natural environment the timing of biologic phases is controlled by genetic inheritance and variation of light conditions of the environment. Phase-shifts of the circadian rhythm may be inhibited completely by affecting RNA synthesis. Also, the various phase cycles may become disoriented in time and not occur in concert. This would also destroy the neatly timed rhythmic cycle. It would appear, then, that the rhythm of life, so to speak, is under the control of internal and external stimuli, although neither may be positively proved by studies at hand to date. The implication of RNA in the timing mechanism suggests many external stimuli such as x-irradiation, certain antibiotics (i.e., chloromphenicol, actinomycin-D) and ultra-violet irradiation. Endogenous hormones, neurosecretory mechanisms, and enzymes, poorly identified, may alter the biologic clock.

It should be noted at this point that there are many who disagree as to the effect of extraneous forces (as barometric pressure, geomagnetic fields, cosmic rays and the like) and state categorically that frequencies of all biologic clocks are genetically guided. They claim the residual periodic variables function after elimination of obvious external factors.

There is a definite synchronization of rhythms within the same species. This is thought advantageous. Rhythmicity is characteristic of nature. No living thing is static.

Early in this century, it was shown that certain elemental marine organisms showed cycles of behaviour with the same frequency as that of the tides in the areas where they had been collected. One observer noted that ancestors of all present-day living things were subjected to the pull of the tides and that this becomes a sort of built-in clock to our daily rhythms. Breeding rhythms definitely occur in certain plants and animals, especially marine life, and there are those who hold that this also occurs in human childbirth. Caspers reviewed the alleged and observed correlations between lunar rhythm and menstruation rate, distribution of births, urine secretion and color vision changes in man. No conclusions could be drawn of any statistical significance. Bats have been show to have a seasonal change in size of their testes, which increase from January to June and decrease in size from October to December. Tropical climates eliminate the so-called breeding season in animals but the cycle goes on in a different time cycle nevertheless.

Halberg described, quantitatively, eosinophile rhythms in terms of a peak in the morning and low at night. Reversal of light source here will reverse the eosinophile rhythm. In this same regard, there have also been described rhythms in numbers of thrombocytes, leucocytes, and red blood cells as well as cholesterol and blood sugar, blood chloride, pulse rate, blood pressure. Halberg also reported blood eosinophile and rectal temperature rhythms persisting in man after subtotal removal of a cerebral hemisphere. The regular variations in sleepiness, temperature, and urinary flow, according to Mills, might be related to an habitual hypothalamic rhythm ultimately derived from external rhythms. Kleitman noted the remarkable persistence of normal temperature and urinary rhythms in night workers in urban communities, as well as diurnal fluctuations of temperature, sleepiness, wakefulness.

Halberg feels that the adrenal cycle describes processes occurring in preparation for daily activities instead of being a reflex reaction or feedback response to them. Kleitman felt that the rhythm of body temperature appeared to be affected by the environment. Potassium excretion was thought, however, determined by an intrinsic twenty-four mechanism.

The search for a center controlling diurnal rhythms has led many investigators far afield but to date no one area has been found. Hypophysectomy and thyroidectomy according to Richter, may disturb the rhythm but not eliminate it. This is also true in subtotal gastrectomy.

Lewis and Lobban conclude that sleep rhythm is controlled by an area of the hypothalamus (not further localized) as are retention of water, chlorides, and body temperature. Potassium excretion on the other hand is under the aegis of the adrenal cortex. There must, obviously, be no single biologic clock system, even though Swan has suggested that probably all cells possess an inherent twenty-four hour rhythm. Halberg described the number of eosinophiles in the blood reflecting the course of the adrenal cycle. The adrenal gland hormones diminish the number

of eosinophiles, the chief basis of our present-day "Thorn" test. Removal of the adrenal gland abolishes eosinophilic rhythm as well as glycogen rhythm in the liver according to Agren. The central nervous system is not required for adrenal rhythm as evidenced by the cycle being found in brainless animals, as noted by Halberg.

The biologic clock may be "set" by light-dark cycles or by temperature controls, as alluded to above, but all living matter display incessant movement to a degree in space and time. The endogenous rhythm will correlate with the external rhythm but persist even though the external stimuli are removed. There is a definite time sense of plants and animals.

Seasonal rhythms occurring in pathology, are well known and add up to a seasonal mortality rhythm. Many infectious diseases have seasonal peaks. Children are thought to grow rhythmically with a peak in spring. Even certain disease states will have a definite rhythm. An example is given by Baker who noted his patient to have maximum knee-joint swelling every seven days in intermittent hydrathrosis (secondary to brucellosis). Periodicity and rhythmicity of peptic ulcer disease and to a lesser extent, chronic ulcerative colitis exacerbations and diseases of atopy (Spring-Fall) are well known. Also it is temptingly intriguing to include here the various conditions which lead to antibody formation of which Hashimoto's disease of the thyroid may be considered a prototype. Less well known but also recorded, Leonard showed freedom of symptoms in his patient with Parkinson's disease at certain times each day. The 280 day cycle of pregnancy would also be included here.

The periodic swelling and regression of the lymph glands and, presumably, corresponding fluctuations in numbers of lymphocytes, as well as the characteristic temperature variations, are noted in the Pel-Ebstein type of Hodgkin's disease. There is also evidence to show the presence of a biological clock in varied phases of catatonic-schizophrenia according to Richter. The periodic illnesses of Reimann may also be cited here.

Since we now have laboratory conditions available to permit fairly accurate measurement of circadian rhythmical oscillations, we are able to recognize organisms indicating and making use of the time of day by their biological clocks. This has intensified interest in time-measurement in physiopathological disturbances, as mentioned earlier. It might be noted, especially, on space voyages where test subjects have great difficulty adjusting to rapidly changing day-night cycles which deviate too much from their usual twenty-four hour cycle or on long modern submarine trips where the day-night cycle rarely changes and adjustment is necessary and often difficult.

Like it or not, we must accept rhythms in our diagnostic armamentarium if we are to succeed in early diagnosis. Difficulties in establishing true basal states for various sized individuals of both sexes and the several races in the setting of varied seasonal fluctuations tends to cause serious re-evaluation of the so-called "tables of normal values" found in most textbooks and other sources. An understanding of biologic rhythmic patterns as plants and animals adapt themselves to the spatial conditions of their environment will obviously ultimately allow optimum scheduling for surgery, giving of medications, and perhaps most important of

all, may open the doors to development of far more sophisticated tests for evaluating physiologic phenomena than any now on the horizon. The study of biologic rhythms is a young interdisciplinary science, leaning heavily on the physical sciences. Many answers await the unprejudiced, inquiring mind of medicine. ROBERT EDGAR MITCHELL, JR., M.D.

The Chiefs versus the Indians

THE GUEST EDITORIAL in this issue of the Virginia Medical Monthly deals mainly with the need for representation of hospital staff nurses on the board of the Joint Commission on Hospital Accreditation and the appointment of the directors of nurses to the boards of directors of community hospitals. The recommendation is also made that a nurse consultant should participate in hospital planning and hospital staff nurses should be adequately represented on national, state, and local committees that deal with health care. In order to put these requests in proper perspective physicians must recall that hospital staff nurses are the backbone of American nursing and these are the nurses we rely upon to care for the ill.

The writer stated that approximately 70% of all nurses fall into the category of hospital staff nurses. Despite this preponderance, the nursing educators have increasingly taken over the role of representing the nursing profession in America. The writer of this guest editorial asked if it is "not true that today there is more understanding of nursing problems between the hospital staff nurses, physicians and administrators than there is between the hospital staff nurses and nursing educators?" The inference is that nursing educators, and especially those associated with the medical schools of this country, exercise too much influence, and not always for the better, on present-day nursing. Many of their policies are not directed toward providing the additional nurses so sorely needed. The educators are primarily interested in training and furnishing the chiefs but there appears to be relatively little concern as to whether the chiefs, in turn, provide an adequate number of Indians to staff the hospitals and care for the sick.

A new Nursing Practice Act for the State of Virginia was prepared recently with the intention of introducing this in the present General Assembly. The proposed Virginia State Board of Nursing would be composed of five registered professional nurses and one practical nurse. In order to be eligible for appointment to the Board the professional nurses would have to "hold a graduate degree" and have "had at least five years experience in nursing service administartion or in administration or teaching in an educational program."

In the Virginia Nurses Association annual convention last October the members took a long look at the proposed Nursing Practice Act. The VNA decided that the present Code of Virginia that deals with nursing should be retained for the present and the matter has been postponed until 1970. The decision appears to have been a wise one and we hope that more trained professional nurses will become available before the nursing Code is altered for the worse. H. J. W.

News . . . .

Calendar of Events

Medical Seminar—A Continuing Education Presentation of the University of Virginia School of Medicine—Hot Springs—February 1-3, 1968.

Stoneburner Lecture Series on Nephrology—Medical College of Virginia—Richmond—February 22-23, 1968.

Virginia Chapter, American Academy of Pediatrics and the Virginia Pediatric Society—Annual Meeting—Williamsburg Inn and Lodge—Williamsburg—February 23-24, 1968.

AMPAC National Workshop—Sheraton-Park Hotel—Washington, D. C.—March 9-10, 1968.

Sectional Meeting for Physicians and Nurses—Sponsored by American College of Surgeons—Williamsburg—March 11-13, 1968.

Clinical Cardiology—19th Annual Post Graduate Day Program of Roanoke Memorial Hospital—Roanoke—March 21-22, 1968.

Second National Congress on Socio-Economics of Health Care—The Palmer House—Chicago, Illinois—March 22-23, 1968.

Southeastern Surgical Congress—Annual Meeting—Sheraton Park Hotel—Washington, D. C.—March 25-28, 1968.

21st National Conference on Rural Health—Olympic Hotel—Seattle, Washington—March 29-30, 1968.

Second National Congress on Medical Ethics—Drake Hotel—Chicago, Illinois—March 30-31, 1968.

Annual Clinical Conference—Louise Obici Memorial Hospital—Suffolk—April 3, 1968.

Annual Joint Cardiac Symposium—Sponsored by Heart Association of Northern Virginia and Washington Heart Association—Marriott Twin Bridges Motor Hotel—Arlington—April 10, 1968.

Cardiovascular Responses to Anesthesia—Fifth Annual Spring Symposium of Virginia Society of Anesthesiologists—Sheraton Motor Inn—Richmond—April 19-21, 1968.

Virginia Academy of General Practice—Annual Scientific Assembly—The Chamberlin—Fort Monroe—May 9-12, 1968.

Seaboard Medical Association—Annual Meeting—Nags Head, North Carolina—June 21-23, 1968.

9th Annual Cardiovascular Symposium—Sponsored by Tidewater Heart Association and Council on Clinical Cardiology, American Heart Association—Lake Wright Motel, Virginia Beach—September 6-7, 1968.

New Members.

The following members were received into The Medical Society of Virginia during the month of November, making a total membership of 3,461:

Michel Laron Barringer, M.D., Richmond

Stephen Joseph Bednar, M.D., Washington, D. C.

James Robert Brunk, M.D., Harrisonburg

James Ray Callison, M.D., Richmond

Miguel Ramon Calzadilla, M.D., Alexandria

Charles David Cooper, M.D., Alexandria

James Gordon Creveling, Jr., M.D., Richmond
Alonzo Watt Douglas, M.D., Newport News
William Jackson Frable, M.D., Richmond
Robert Edward Gardner, M.D., Rocky Mount
Phyllis Anne Huene, M.D., Falls Church
Bruce Jeremy Maitland Innes, M.D., Norfolk
Robert Gentry Kendall, M.D., Winchester
James David Kitchin, III, M.D., Charlottesville
Harold Brian Landes, M.D., Winchester
Louis Benedick Massad, M.D., Fredericksburg
Francis Stanford Massie, M.D., Richmond
Robert Thomas Mosby, Jr., M.D., Virginia Beach
Alonzo Harrison Myers, Jr., M.D., Roanoke
Santiago Nunez, M.D., Richmond
Thomas George Peters, M.D., Ladysmith
Richard Francis Sappington, Jr., M.D., Alexandria
Leroy Fleming Smith, Jr., M.D., Alexandria
Yasuaki Takagi, M.D., Fairfax
Jon Bunde Tingelstad, M.D., Richmond
James Stuart Wolf, M.D., Richmond
Edward Albert Zakaib, M.D., Alexandria

Northern Neck Medical Association.

Dr. Harold E. Sisson, Warsaw, has been elected president of this Association. Dr. J. Motley Booker, Lottsburg, is president-elect; Dr. Louis D. Parham, Montross, vice-president; and Dr. John H. Tyler, White Stone, secretary-treasurer.

Dr. Robert M. Miskimon,

Richmond, has been elected to the Board of Directors of the Medical Education Life Fund, Incorporated. He is vice president and medical director of the Fidelity Bankers Life Insurance Company of Richmond.

Memorial to Dr. Woodhouse.

The Virginia Beach Medical Society and friends of the late Dr. Robert Woodside Woodhouse, Jr., have established a memorial fund in his memory. This fund will be used for medical education purposes possibly in the form of scholarships. Contributions may be sent to the Memorial Fund in care of the Virginia Beach Medical Society, 1060 First Colonial Road, Virginia Beach.

Stoneburner Lecture.

The 21st Annual Stoneburner Lecture Series will be held at the Medical College of Virginia, February 22-23. The subject for discussion will be Nephrology—Emphasis on Acid-Base Balance, Structural and Functional Renal Disorders, and the Problems of the Management of Acute and Chronic Renal Disease.

Maurice B. Strauss, M.D., Boston, will be the Special Guest Lecturer, and other members of the guest faculty are: Frederic Barttner, M.D., Bethesda, Maryland; William Falls, M.D., Richmond; Lee W. Henderson, M.D., Philadelphia; Robert H. Heptinstall, M.D., Baltimore; Calvin M. Kunin, M.D., Charlottesville; David Lauler, M.D., Boston; Lott Page, M.D., Boston; John H. Peters, M.D., Boston; Victor E. Pollak, M.D., Chicago; George Schreiner, M.D., Washington, D. C.; and Roscoe R. Robinson, M.D., Durham, North Carolina. Members of the Medical College of Virginia faculty are: Newton C. Brackett, M.D., David M. Hume, M.D., Russell E. Randall, M.D., and John G. Setter, M.D.

A block of rooms has been reserved for registrants at the John Marshall Hotel and reservations should be requested directly from the Hotel.

Associates Wanted.

Generalist or internist to associate with busy fifty year old generalist. Richmond environs. Excellent hospital facilities. Are you tired of solo practice? Join me in a

well-run practice that offers outstanding professional and financial rewards. Work opposite me. No investment. Expense sharing arrangement. Write #80, care Virginia Medical Monthly, 4205 Dover Road, Richmond, Virginia 23221. (*Adv.*)

Wanted.

Physician, G. P. level—chronic diseases. 153-bed service in 941 bed GM&S V.A. Hospital, Richmond, Virginia. Salary: $12,873-$19,813, dependent upon qualifications. Citizenship or immigrant status; licensure any state required. Annual leave 30 days a year; excellent retirement; health and life insurance plans; and other benefits. Nondiscrimination in employment. Write Chief of Staff, Veterans Administration Hospital, Richmond, Virginia 23219. (*Adv.*)

Obituary....

Dr. Robert Woodside Woodhouse, Jr.,

Virginia Beach, died on November 4, following a heart attack. He was eighty-two years of age and the last of the horse-and-buggy doctors in the Virginia Beach area. Dr. Woodhouse graduated from the former University College of Medicine, Richmond, in 1913. He had practiced at Virginia Beach since his graduation and for many years was the only general practitioner there. Dr. Woodhouse was named the First Citizen of Virginia Beach in 1955 by the Junior Chamber of Commerce. He had been a member of The Medical Society of Virginia for fifty-four years.

A son, Dr. Woodhouse, III, of Richmond, survives him.

Dr. Charles Perry Howze,

Charlottesville, died October 26. He was seventy-two years of age and received his medical degree from the University of Virginia in 1918. Dr. Howze had been a member of The Medical Society of Virginia since 1954.

His wife and two sons survive him.

Dr. Haddon Christopher Alexander, Jr.,

Farmville, died November 4, at the age of seventy. He was a graduate of the Medical College of Virginia, class of 1926. Dr. Alexander was a member of the staff of the Southside Community Hospital. He was a Mason and a member of the local Lions Club. Dr. Alexander had been a member of The Medical Society of Virginia since 1928.

His wife and a son survive him.

Dr. Reynold Clinton Siersema,

Richmond, died November 27, after a long illness. He was sixty-six years of age and a graduate of the Medical College of Virginia in 1934. Dr. Siersema was assistant professor of clinical surgery at the Medical College of Virginia. He was a former track coach and professor of physical education at the College of William and Mary and had been chairman of the physical education department and coach of athletics at Juniata College in Pennsylvania before he began his medical studies. Dr. Siersema was a lieutenant colonel in the Army during World War II and was assistant chief of surgery for the Eighth Evacuation Hospital and was awarded the Bronze Star. He had been a member of The Medical Society of Virginia for thirty years.

His wife and a son survive him.

Dr. Scherer.

Dr. John Hamilton Scherer, pathologist and beloved member of the Richmond Academy of Medicine, died in Richmond on May 18, 1967, following a long illness. His widow and two children survive.

Dr. Scherer was born in Marion, on October 27, 1899. He graduated from Chamberlayne's School in Richmond in 1917 and following this he attended the University of Virginia. From 1921 to 1927 he was a reporter for the Asociated Press, based in Richmond. He then returned to the University of Virginia and graduated in medicine in 1931. For the next five years he trained in pathology and served as Instructor in Pathology at the University of Virginia. During this period he developed a keen interest in hematology, a field in which he excelled throughout his career. In 1936 Dr. Scherer was appointed Director of Clinical Laboratories at the Medical College of Virginia, and he held this position until he entered private practice in 1943. During this period he also served as Coroner for the City of Richmond. During World War II he was sent by the government to the Tulane School of Tropical Medicine and to Central America to study tropical diseases.

Dr. Scherer was a leader in the field of blood banking during its period of rapid expansion following World War II and in 1947 he made a most significant contribution to the Richmond community when he established the Virginia Blood Bank. This was the first community blood bank in this area and throughout the years has given outstanding service to the hospitals and citizens of Richmond.

Dr. Scherer served as Associate Professor of Pediatrics in Hematology at the Medical College of Virginia, Medical Consultant to the Atlantic Coast Line Railroad, Medical Director of the Virginia Blood Bank, and Pathologist for St. Luke's Hospital, Grace Hospital, Retreat for the Sick, and Westbrook Psychiatric Hospital. He was a diplomate of the American Board of Pathology, a Fellow of the College of American Pathologists, a Fellow of the American Society of Clinical Pathologists, and a member of the American Association of Blood Banks, the American Medical Association, the Southern Medical Association and The Medical Society of Virginia.

Dr. Scherer was warm, disarming and charming, and a loyal friend and colleague. He was dedicated to his profession and was a willing and patient teacher. He was a generous and kind man, in his family life, to his friends, employees, associates, and especially to those in need. His intellectual integrity was of the highest order; so also were his contributions to his community and his profession. Whenever principles dear to him were threatened, Dr. Scherer would rise to their defense and fight, fairly but without compromise, and always as a gentleman. Throughout his career his prime concern was always for the welfare of the patient.

Be It Resolved: That the Richmond Academy of Medicine enter into its minutes those remembrances of Dr. Scherer and that a copy be sent to the Virginia Medical Monthly and to his family.

Richard W. Fowlkes, M.D.
Edwin L. Kendig, Jr., M.D.
John L. Thornton, M.D.

Dr. Hurt.

Dr. George William Hurt, obstetrician and gynecologist, was killed in an aircraft accident on October 2, 1967. He was forty-five years of age.

Dr. Hurt graduated from Hampden-Sydney College in 1943 and received his medical degree from the Medical College of Virginia in 1946. After completing his internship at the Medical College of Virginia, he served two years as a medical officer in the United States Navy. He was honorably discharged in 1949 and spent a year doing post-graduate work in obstetrics and gynecology at the Graduate Hospital of the University of Pennsylvania. In 1950, he went back to the Medical College of Virginia where he completed a residency in obstetrics and gynecology. Part of this residency was done at the Johns Hopkins Hospital. In 1953, Dr. Hurt returned to Roanoke to go into private practice with his father, Dr. George Samuel Hurt.

He was a Diplomate of the American Board of Obstetrics and Gynecology and a Fellow in the American College of Obstetricians and Gynecologists. He was one of the founders of the Roanoke Valley Association of Obstetricians and Gynecologists and a founding member of the South Central Obstetrics and Gynecologic Society of which he was secretary at the time of his death. He was also an active member of the South Atlantic Association of Obstetricians and Gynecologists, The H. Hudnall Ware, Jr., Society, The Medical Society of Virginia, and the Roanoke Academy of Medicine. He was the first Chief of Obstetrics and Gynecology at the Community Hospital of Roanoke Valley.

Dr. Hurt was a member of Kappa Alpha Order, Phi Chi Medical Fraternity and the Shenandoah Club of Roanoke.

He was a Deacon in Calvary Baptist Church and served on the Board of Directors of the Young Men's Christian Association. He was also a member of the Rotary Club.

"Doctor Billy" was proud of his heritage and his profession. He was devoted to his family. He was conservative in his beliefs but radical in his fight for what he believed. He gave freely and generously of his time and his means to his many civic interests. He was a fine physician, an outstanding teacher, a Christian gentleman, and loyal to his friends.

He is survived by his wife, a daughter, a son, his parents, a sister and a brother.

His untimely death leaves a void that cannot be filled. Although this community is better for his having lived here, he will be deeply missed.

Therefore Be It Resolved a record of his passing and our loss be made a permanent part of the minutes of the Roanoke Academy of Medicine and copies be sent to The Medical Society of Virginia and to his wife.

W. W. S. Butler, M.D.
David P. Minichan, Jr., M.D.
David W. Branch, M.D.

Dr. Wilkins.

On August 8, 1967, Dr. William Beverley Wilkins was called to rest on Cozumel Island, Mexico, after 35 years of devoted service in the practice of medicine.

He was born in Alexandria, on August 8, 1904. He attended public schools in Strasburg, Alexandria and Charlottesville. He attended the University of Virginia for his undergraduate work and was graduated from the University of Virginia Medical School in 1932. He interned at the Lloyd Nolan Hospital in Birmingham, Alabama, and later took postgraduate studies in cardiology at Massachusetts General Hospital and the University of Michigan.

He served as a flight surgeon in the Air Force from 1942 to 1946. During his service with the Air Force he sustained a heart attack which somewhat limited his activities, but in no way curtailed his zeal and pursuit of excellence in medicine.

During his earlier years, his hobbies were swimming, fishing, hunting and boating. He was a conservationist of nature and was fascinated by outdoor life and the natural beauty of unspoiled virgin land. It was the latter interest which influenced him to retire on the lovely Cozumel Island in Mexico.

Indeed there was never a more devoted scholar of medicine. He believed in and lived by the oath of Hippocrates. His inquisitive and searching mind was forever seeking the hidden secrets of medicine and nature.

His keenness and wit made him a much sought-after social companion. He was a gracious gentleman who valued friendship more than material things.

Dr. Wilkins leaves his wife and many friends and colleagues the fond memory of a warm personality and a conscientious physician.

Be It Therefore Resolved that this brief resume of the useful life of Dr. William B. Wilkins be spread in the minutes of the Medical Staff of the Circle Terrace Hospital and that a copy be sent to his family and the Virginia Medical Monthly.

Dr. Lee.

Dr. Elizabeth Lee died October 3, 1967. Elizabeth Saunders Lee was born and raised in Blacksburg. She was an honor graduate of Hollins, class of 1926. The following year she taught music at St. Catherine's school in Richmond. In 1927-28 she attended V.P.I. to complete her pre-medical requirements. She graduated in 1932 from the University of Virginia School of Medicine, where she was elected to membership in Alpha Omega Alpha and Phi Beta Kappa. At the University of Virginia Hospital she interned for two years; took a year in pediatrics at St. Luke's Hospital in New York City, and a year of obstetrics at Johns Hopkins. She was in general practice in Blacksburg until her marriage to Dr. Henry Lee in 1939. In Roanoke she continued her practice and was part-time physician to Hollins College until 1942—when her husband went into active service in the Army. While he was overseas she and their young family moved to Blacksburg. Upon his return, they moved back to Roanoke. In 1948 she became instructor in hygiene and college physician at Hollins.

She will be remembered by her friends, associates, and students for her crisp humor and her devotion to medicine, and to her husband and four children. One son is a lawyer; another is in law school; a third son is still in service having just returned from Vietnam. Her daughter is in Medical School at the University of Virginia.

Be It Resolved that this notice be made a part of the minutes of this meeting; and that a copy be sent to the Virginia Medical Monthly; and to her family with an expression of deepest sympathy from the Roanoke Academy of Medicine.

C. D. Smith, M.D.
Edgar M. Weaver, M.D.
R. Earle Glendy, M.D.
Margaret M. Glendy, M.D., *Chairman*

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Frank M. Blanton, M.D.
William W. Martin, Jr., M.D.

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Spotswood Robins, M.D.
David C. Forrest, M.D.
Joseph C. Parker, M.D.
William M. Oppenhimer, M.D.

Orthopedics:
Beverley B. Clary, M.D.
James B. Dalton, Jr., M.D.
Franklin P. Watkins, M.D.

Pediatrics:
Edward G. Davis, Jr., M.D.
Thomas P. Overton, M.D.
Albert S. Thompson, Jr., M.D.
Edward J. Wiley, M.D.

Ophthalmology, Otolaryngology:
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J. Warren Montague, M.D.

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William B. Moncure, M.D.
Heth Owen, Jr., M.D.

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Carrington Williams, M.D.
Richard A. Michaux, M.D.
Carrington Williams, Jr., M.D.
Armistead M. Williams, M.D.
James R. Darden, Jr., M.D.

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Frank Pole, M.D.
Harry S. Rowland, Jr., M.D.

Oral Surgery:
Guy R. Harrison, D.D.S.

Physical Therapy
Marilyn B. Morrison, R.P.T.

Plastic Surgery:
Hunter S. Jackson, M.D.

Roentgenology and Radiology:
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William C. Barr, M.D.
James W. Proffitt, M.D.

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Director:
Charles C. Hough

When the talk turns to oral contraceptives, it makes medical sense to remember low-dose Norinyl®-1.

(norethindrone 1mg ē mestranol 0.05mg)

Turn page for contraindications, precautions and side effects.



Reduction of oral contraceptive dosage to the lowest effective levels is a well-accepted principle of conservative medical practice. In keeping with this view, Norinyl is now also available as Norinyl-1, containing exactly one half the previous dosage of norethindrone and mestranol. Clinical experience has established that effective fertility control can be achieved with the same degree of reliability and safety with new Norinyl-1 when taken as directed.

What about switching patients from higher dosage forms?

In transferring patients to low-dose Norinyl-1 from higher-dosage oral contraceptives, some breakthrough bleeding may occur in the early cycles. In the majority of cases the bleeding episode is mild and self-limited. The long-term advantages of the lower dosage form should be weighed against the inconvenience of possible breakthrough bleeding in the individual patient.

Prescribing Information

Contraindications: Patients with any symptoms or history of thrombophlebitis, pulmonary embolism, liver dysfunction or disease, carcinoma of breast or genital organs, or undiagnosed vaginal bleeding.

Warnings: Discontinue medication pending examination if there is sudden partial or complete loss of vision, proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions, medication should be withdrawn. The safety of Norinyl-1 in pregnancy has not been demonstrated. If a patient misses two consecutive periods, pregnancy should be ruled out before continuing the medication. If she has not adhered to the prescribed schedule, pregnancy should be considered at the first missed period. Active ingredients of oral contraceptives have been detected in the milk of mothers who received these drugs; the significance to infants has not been determined.

Precautions: Pretreatment physical should include examination of the breasts and pelvic organs, as well as a Papanicolaou smear. If endocrine or liver function tests are abnormal during therapy, repeat tests are recommended after the drug has been withdrawn for two months. Following administration of drug, preexisting uterine fibromyomata may increase in size. Careful observation and caution are required for patients with symptoms or history of epilepsy, migraine, asthma, cardiac or renal dysfunction, cerebrovascular accident, psychic depression, and diabetes. In cases of undiagnosed vaginal bleeding, adequate diagnostic measures are indicated. Possible long-term effects of the drug on pituitary, ovarian, adrenal, hepatic or uterine function must await further studies. The physician should be alert to the earliest manifestations of thrombophlebitis and pulmonary embolism. The drug should be used judiciously in those young patients in whom bone growth is not complete. The age of the patient constitutes no absolute limiting factor, although treatment with Norinyl-1 may mask symptoms of the climacteric. The pathologist should be advised of Norinyl-1 therapy when relevant specimens are submitted.

Side Effects: The following have been observed with varying incidence in patients receiving oral contraceptives: nausea, vomiting, gastrointestinal symptoms, breakthrough bleeding, spotting, change in menstrual flow, amenorrhea, edema, chloasma or melasma, breast changes (tenderness, enlargement and secretion), change in weight (increase or decrease), changes in cervical erosion and cervical secretions, suppression of lactation when given immediately postpartum, cholestatic jaundice, migraine, rash (allergic), rise in blood pressure in susceptible individuals, mental depression. Although the following side effects have been reported in users of oral contraceptives, no cause and effect relationship has been established: anovulation posttreatment, premenstruallike syndrome, changes in libido, changes in appetite, cystitislike syndrome, headache, nervousness, dizziness, fatigue, backache, hirsutism, loss of scalp hair, erythema multiforme, erythema nodosum, hemorrhagic eruption, and itching. The following occurrences have been observed in users of oral contraceptives (a cause and effect relationship has neither been established nor disproved): thrombophlebitis, pulmonary embolism, neuroocular lesions.

The following laboratory tests may be altered by the use of oral contraceptives: increased sulfobromophthalein and other hepatic function tests, coagulation tests (increase in prothrombin, factors VII, VIII, IX and X), thyroid function (increase in PBI and butanol extractable protein-bound iodine and decrease in T^3 values), metyrapone test, pregnanediol determination.

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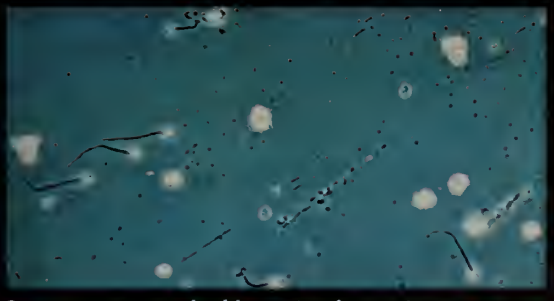
Here's why Norinyl-1 makes medical sense.

The effectiveness of Norinyl-1 as a low-dose oral contraceptive may be explained by its possible multiple action. In addition to its primary action of suppression of ovulation, Norinyl-1 may offer additional protective mechanisms... (1) creation of a cervical mucus that may be hostile to sperm penetration, and (2) development of an endometrium that may be out of phase with nidation. These effects are illustrated below.

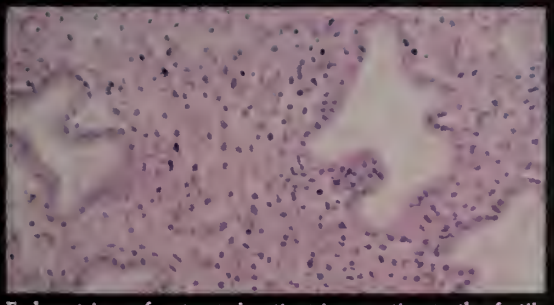
Untreated Patient



Cervical mucus at midcycle is usually thin and watery, with Spinnbarkeit (stretchability) of 15 to 20 cm.



Spermatozoa appear healthy, active, freemoving.

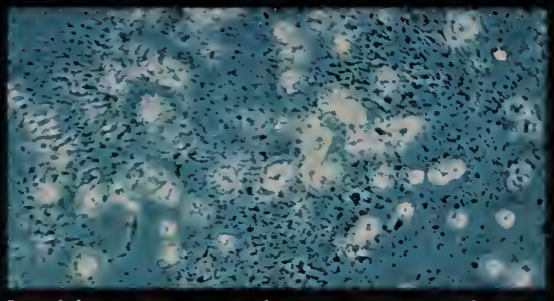


Endometrium of untreated patient is receptive to the fertilized ovum during secretory phase.

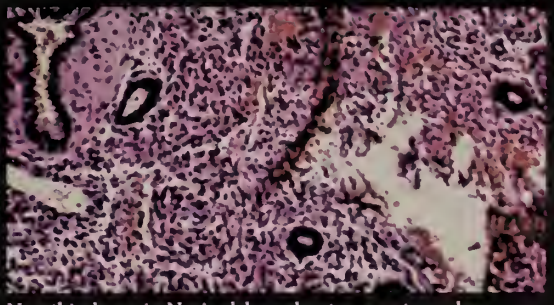
Norinyl-1 Patient



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Turn page for contraindications, precautions and side effects.

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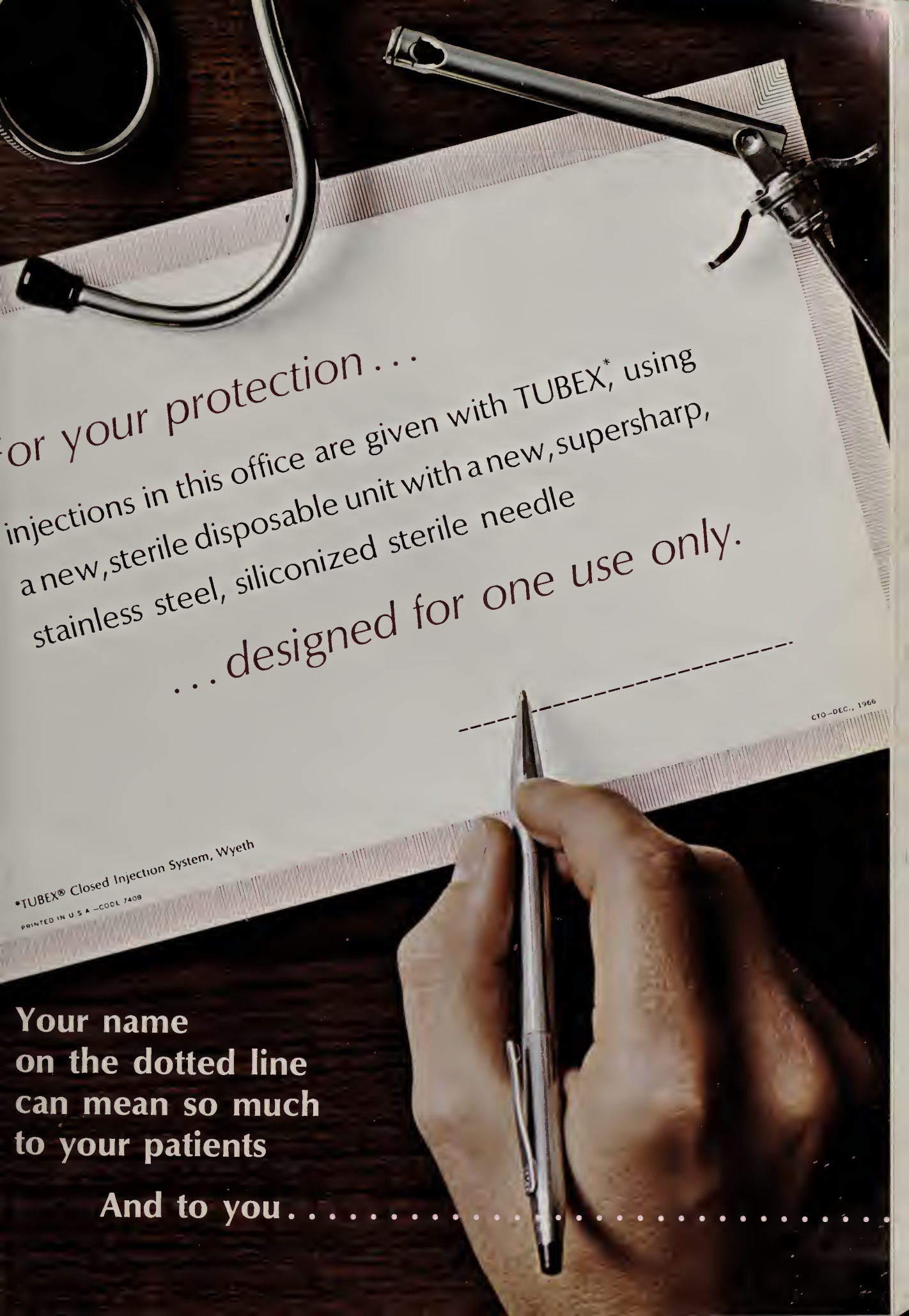
Contraindications: Tuberculous, fungal, and most viral lesions of the skin (including herpes simplex, vaccinia, and varicella). Not for ophthalmic use. Contraindicated in individuals with a history of hypersensitivity to any of the components. *Precautions:* Synalar preparations are virtually nonsensitizing and nonirritating. However, the solution may produce burning or stinging when applied to denuded or fissured areas. In some patients with dry lesions, the solution may increase dryness, scaling or itching. Where severe local infection or systemic infection exists, the use of systemic antibiotics should be considered, based on susceptibility testing. While topical steroids have not been reported to have an adverse effect on pregnancy, the safety of their use on pregnant females has not absolutely been established. Therefore, they should not be used extensively on pregnant patients, in large amounts, or for prolonged periods of time. *Side Effects:* Side effects are uncommon with topical corticosteroids. As with all drugs, however, a few patients may react unfavorably to Synalar under certain conditions. In such cases the agent should be discontinued and appropriate measures taken. *Availability:* Synalar (fluocinolone acetonide) Cream 0.025%—5, 15 and 60 Gm. tubes and 425 Gm. jars. Cream 0.01%—15, 45 and 60 Gm. tubes and 120 Gm. jars. Solution 0.01%—20 and 60 cc. plastic squeeze bottles. Ointment 0.025%—15 and 60 Gm. tubes. Neo-Synalar® (neomycin sulfate 0.5% [0.35% neomycin base], fluocinolone acetonide 0.025%) Cream—5, 15 and 60 Gm. tubes.

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1. Bradley, J. E., *et al.*: J. Pediat. *38*:41 (Jan.) 1951.
2. Bradley, J. E.: Mod. Med. *20*:71 (Oct. 15) 1952.
3. Crunden, A. B., Jr., and Davis, W. A.: Am. J. Obst. & Gynec. *65*:311 (Feb.) 1953.



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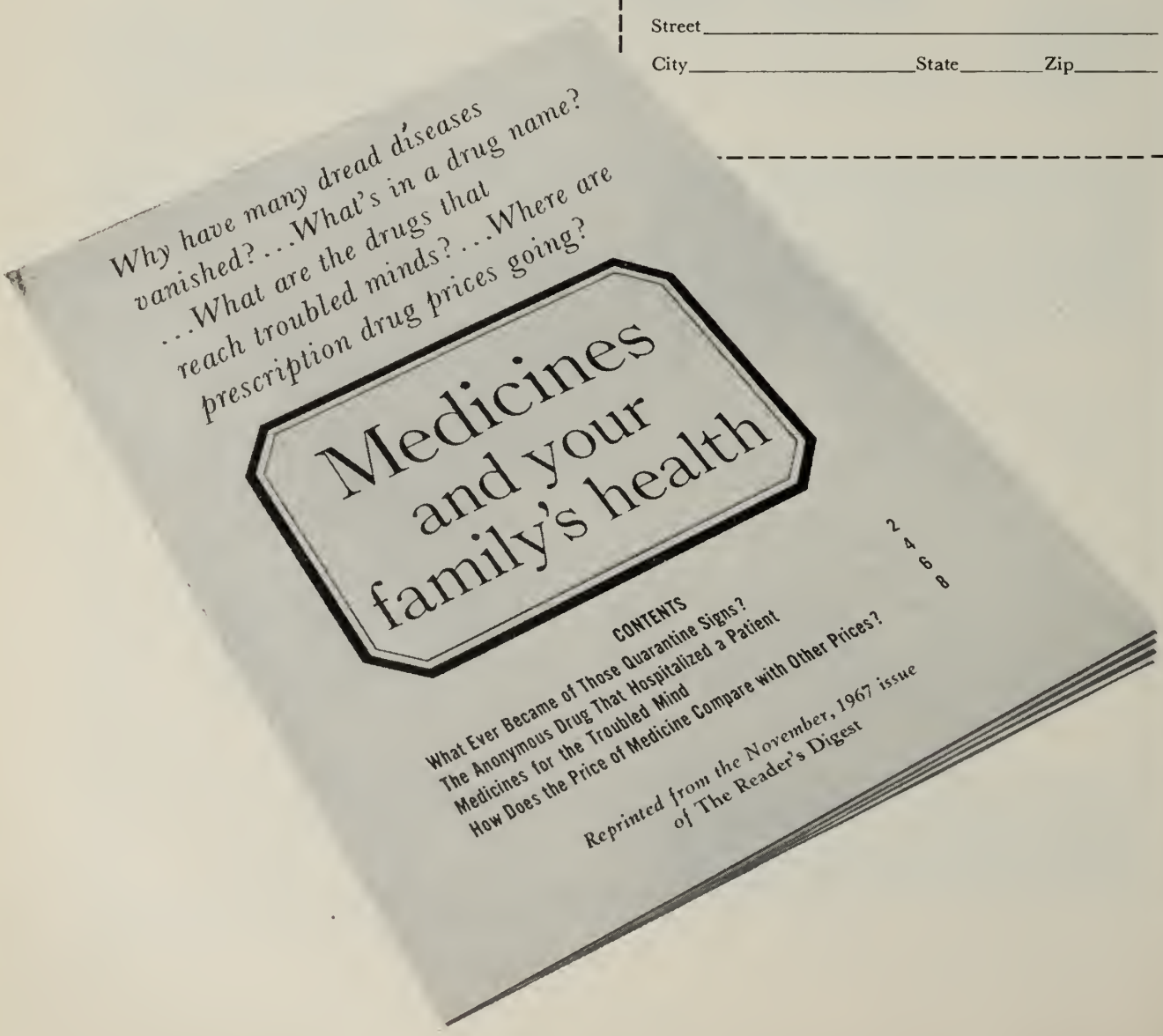
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this issue: the cold in pregnancy...

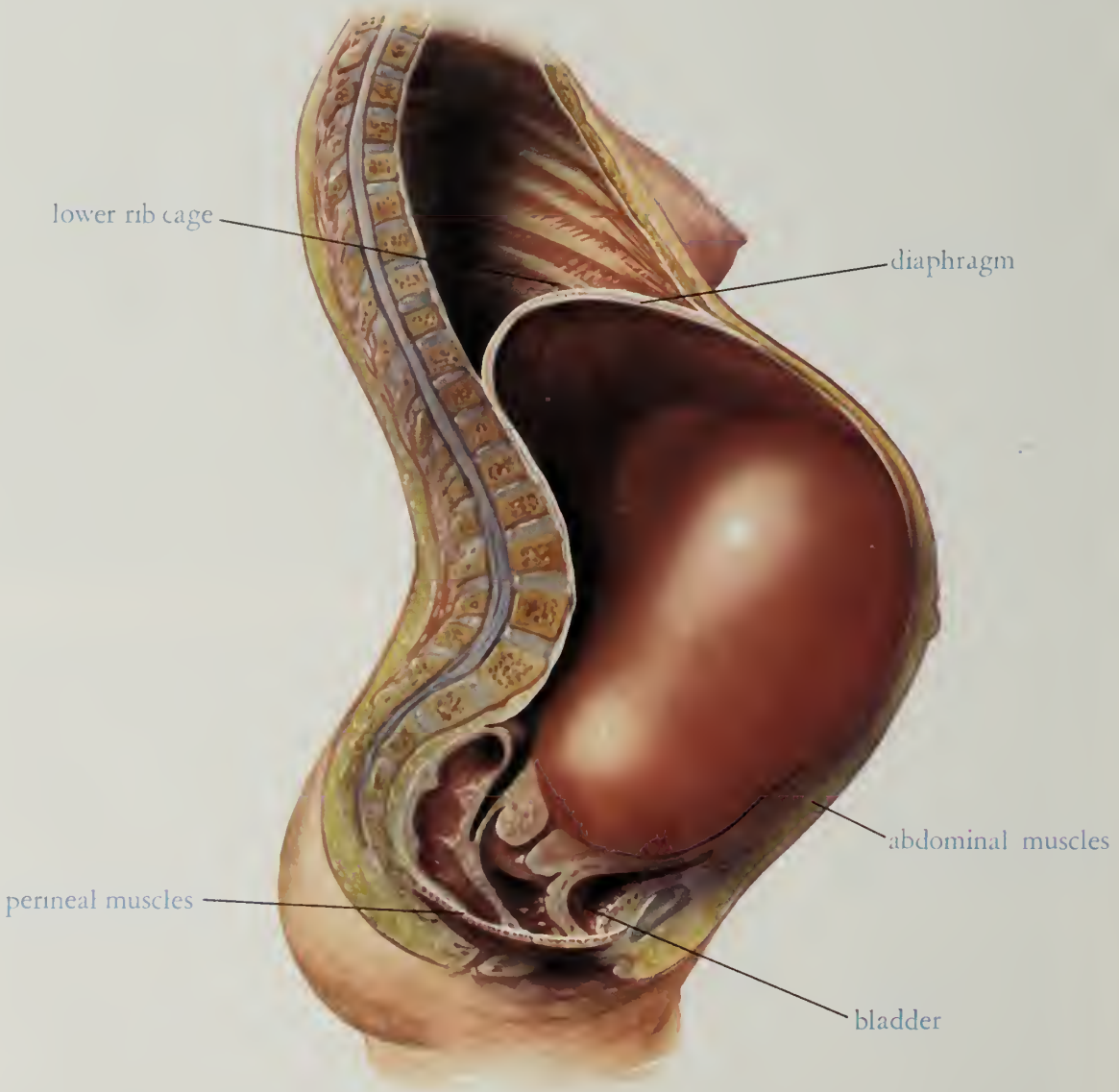
the cold in pregnancy

Frederick W. Goodrich, Jr., M.D.
Senior Obstetrician and Gynecologist, Lawrence and Memorial Hospital, New London, Connecticut

Since pregnancy and the common cold are both ubiquitous, it is not surprising that they often occur at the same time in the same patient. Incidence figures are hard to come by, but the chances are very good that any given pregnant woman will have a cold at some time during the nine months of her gestation. When she does, she will tell you that it is the worst cold she ever had. It seems to last longer and the symptoms are more distressing than they are in the non-pregnant.

For purposes of this discussion, the common cold is considered to be the well-known symptom complex which includes sore throat, stuffy nose, and a cough. Febrile states or extension of the disease process into the lower respiratory tree are not part of the common cold and will not be included in this discussion.

The clinical picture of a cold in pregnancy can be confused by a long-known physiological phenome-



ıon. Kiesselbach's area in the nose (on the anterior ɔart of the nasal septum above the intermaxillary ɔone) becomes engorged, apparently due to hyperemia induced by the increased estrogen level which ıccompanies pregnancy. The amount of congestion ıan vary in degree from woman to woman. Some ıave very little congestion, others will have occaional nosebleeds from this area, still others will ıave symptomatic congestion to the degree that they vill complain of having a "chronic" or constant old.

ess well recognized is the occurrence of this type f hyperemia in any part of the nasopharyngeal ıucosa, again in varying degree. Such swelling ften produces a postnasal drip which, the patient vill state, is present only when she is pregnant. 'atients who do not have symptomatic congestion rdinarily, will find that when they do get a cold, ıe symptoms last much longer than those of a cold sually do. Occasionally, this hyperemia is respon-ible for closure of the medial end of the Eustachian ube; such patients will complain of "plugging" of ıe ears. Inspection of the ear drum will show a epression which confirms the presence of Eusta-hian closure rather than wax in the canal which is ıe patient's diagnosis. Symptoms related to this hysiological congestion are more apt to occur in eavier smokers or those who have a history of llergic rhinitis, just as are the symptoms of the ɔmmon cold. And when the cold does occur in regnancy, the symptoms are worse because of the nderlying congestion.

The pregnant woman with a cold is miserable for ther reasons, dependent somewhat on her parity nd the length of her gestation. As parity increases, ɔ also does the relaxation of the abdominal and erineal musculature. The uterus, lying against a ack abdominal wall, and bearing down on relaxed erineal muscles, acts like a piston when the patient ɔughs, sneezes, or even blows her nose, pushing own on the bladder. Stress incontinence during ɔlds is almost the rule.

s the length of gestation increases, so does the size f the uterus. As it grows, it pushes the abdominal ɔntents above it and elevates the diaphragm. This esults eventually in a lateral displacement of the ɔwer rib cage, often to a point at which the patient vill complain of soreness in this area. If such a

(Concluded on following page)

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patient has a cold, the coughing and sneezing will exaggerate this soreness. The diaphragmatic restriction caused by the enlarging uterus also makes breathing more difficult, particularly when the patient lies down. A cold compounds this difficulty.

Treating a cold in pregnancy requires two additional considerations. The patient should be told that the symptoms may last longer lest she become discouraged, and she should be reassured that the treatment will not damage the fetus. Although there are still some women who seem blissfully ignorant regarding matters of health (and these need to be cautioned not to take medication recommended by friends), most of our maternity patients are aware of the thalidomide tragedy. These women need constant reassurance that the symptoms of a cold can be treated, and that the medication is safe. Fortunately, many of the drugs which are of benefit have been in use long enough and widely enough so that such a statement can be offered. Occasionally one encounters a patient who is so anxious to avoid medication that she is extremely reluctant to take anything at all. Since cold is self-limited, it seems reasonable enough to give such a patient the choice and not to force drugs on her if she is obviously afraid of taking them.

The most distressing symptom of the common cold is the nasal congestion. This can be mitigated by the use of a decongestant which will help reduce the postnasal drip and the coughing caused by it. The kind of a cough which is described as a "tickling" in the throat and which is apt to occur when the patient goes to bed is usually due to nasopharyngeal congestion rather than inflammation in the lower respiratory tract.

There are many cough mixtures available. Most contain an antihistamine, a decongestant, and a cough "suppressant." Should cough suppression be desirable, it is important to remember not to use both a decongestant tablet and such a cough mixture as the patient would be getting a double dose of the decongestant. With some patients, the placebo effect of a cough syrup may be important and a liquid preparation might be substituted for an oral tablet.

For a sore throat, candy drops or lozenges at frequent intervals are soothing. It is not only unnecessary but unwise to use antibiotic lozenges or drops. The use of antibiotics in an uncomplicated cold is contraindicated and should be scrupulously avoided.

In summary, a cold in pregnancy is more severe and longer lasting. The treatment of the symptoms with local and systemic decongestants will make the patient more comfortable.

Apparently the cold is so common in pregnancy that it has received very little attention in the literature. References are almost non-existent and the few which are available add little to the common knowledge, are out-dated, or are not helpful. Thus the usual bibliography is not appended.



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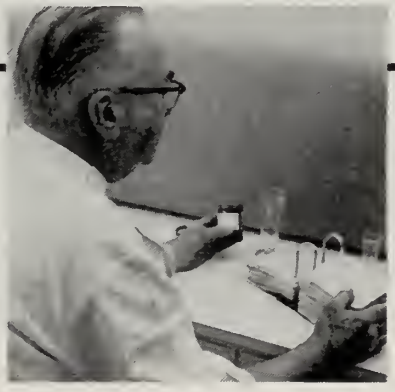
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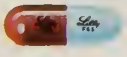
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Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been establisned clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver-function tests advisable during protracted therapy.

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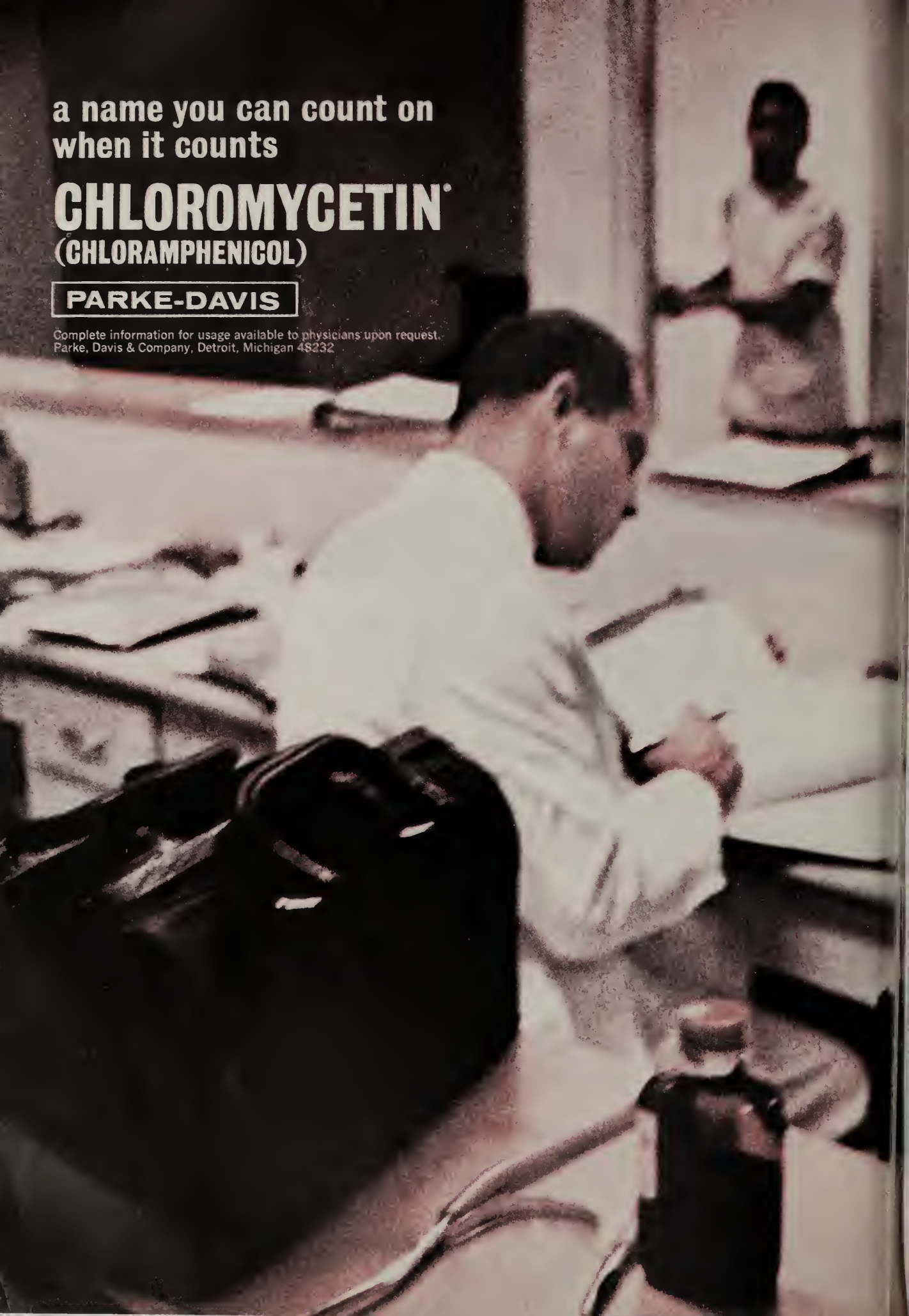
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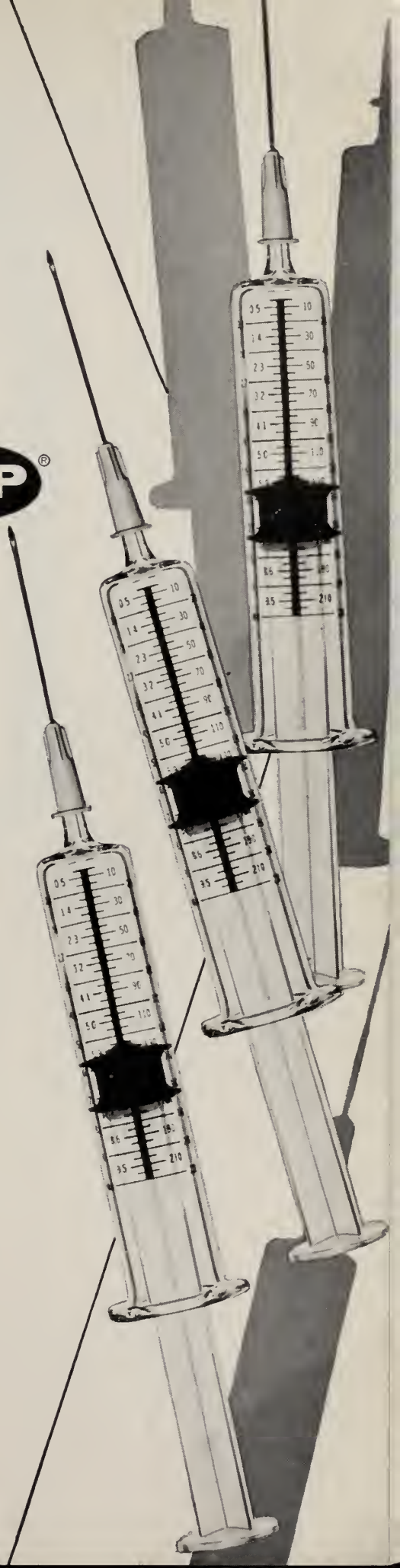
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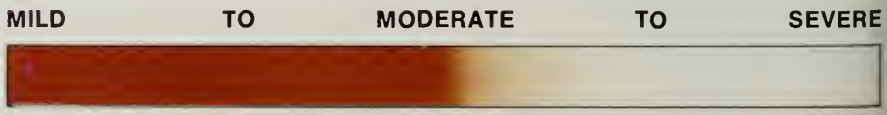
Of course, as with all thiazides, supplemental dietary potassium should also be considered.

Use Enduron as a basic therapy in patients with mild to moderate hypertension. A single 5-mg. tablet each day is ample in most cases.

Once a day, every day

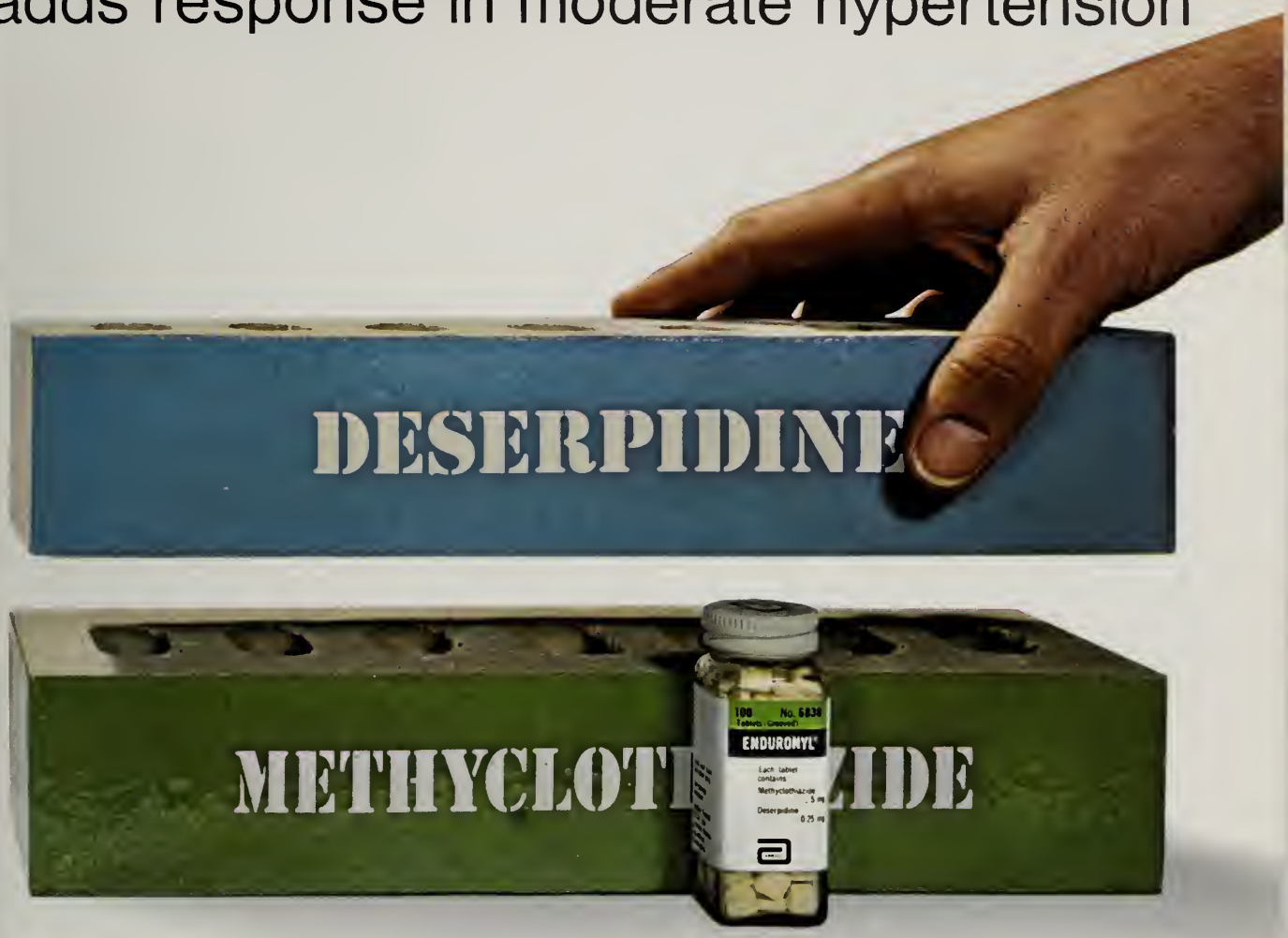
ENDURON

METHYCLOTHIAZIDE



See Brief Summary on final page of advertisement

Enduronyl: Its deserpidine component adds response in moderate hypertension



Less frequent rauwolfia side effects than with reserpine

When you wish to build further response, consider shifting to Enduronyl.

Enduronyl adds a building block of *deserpidine*. This is a purified rauwolfia alkaloid available only from Abbott. It adds good antihypertensive and tranquilizing activity. Yet its incidence of untoward effects, particularly lethargy and depression, is lower than with reserpine.

Enduronyl is available plain or Forte. The latter provides its variation where most helpful, by doubling the deserpidine.

Use Enduronyl for patients throughout the broad range of mild to moderately severe hypertension.

Once a day, every day

ENDURONYL®

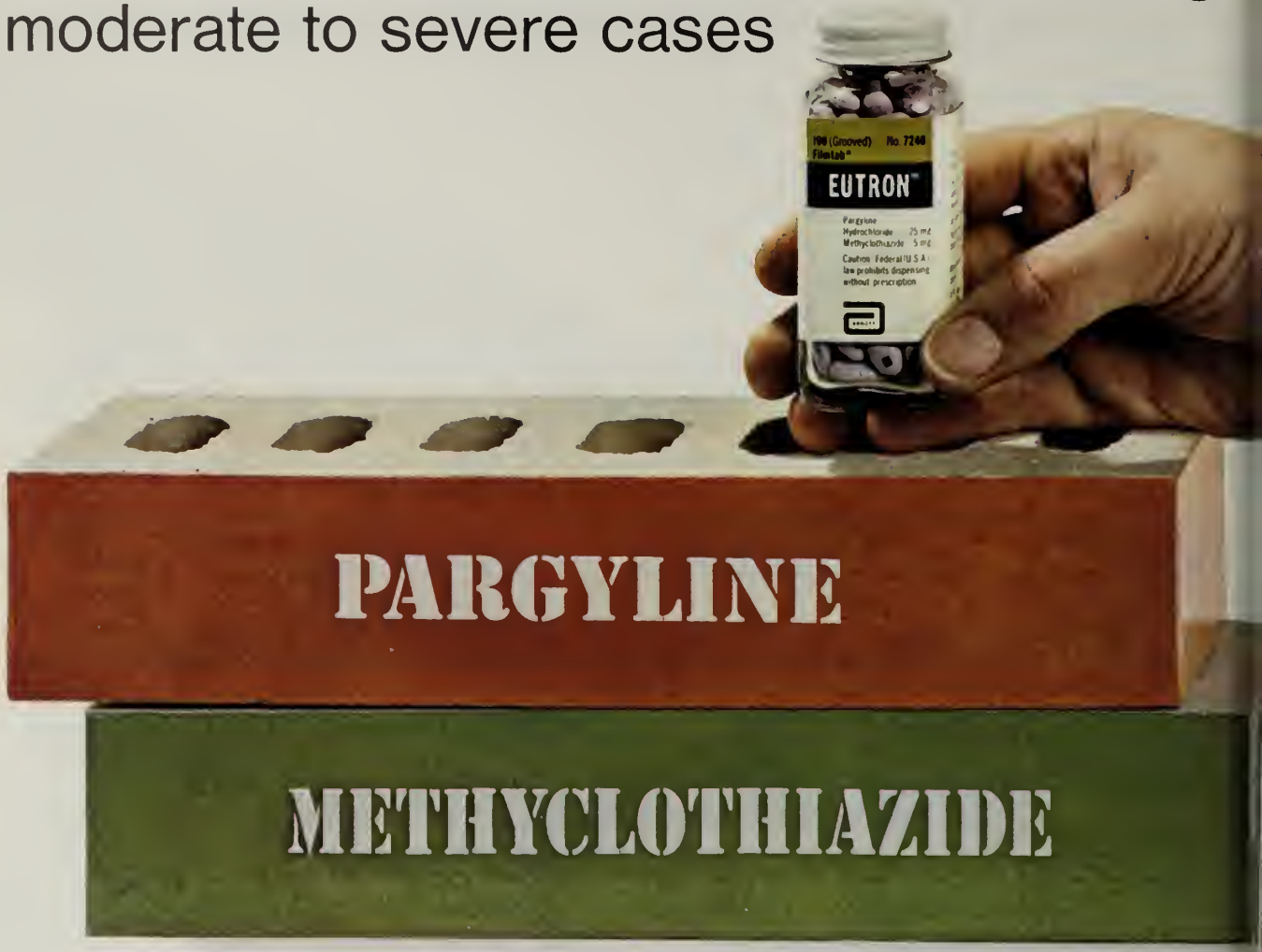
METHYCLOTHIAZIDE 5 mg. with
DESERPIDINE 0.25 mg. or (FORTE) 0.5 mg.

MILD TO MODERATE TO SEVERE



See Brief Summary on final page of advertisement

Eutron: A unique combination for handling moderate to severe cases



Affords almost uniform diastolic reduction in all body positions

Eutron lowers diastolic pressures nearly equally, whether your patient is standing up or lying down.

Thus, in clinical trials, average *standing* diastolic readings were reduced from 112 pre-treatment to 90 post-treatment; *sitting* from 115 to 95; and *recumbent* from 112 to 94.

Note that following Eutron, the diastolic reductions were nearly alike in all three body positions.

Use Eutron for managing your moderate to severe cases. Its building blocks enhance each other; hence lesser doses often suffice.

Once a day, every day

EUTRON™

PARGYLINE HYDROCHLORIDE 25 mg.
with METHYCLOTHIAZIDE 5 mg.

MILD TO MODERATE TO SEVERE



See Brief Summary on final page of advertisement

ENDURON® | ENDURONYL®

METHYCLOTHIAZIDE

Each tablet contains Methyclothiazide 5 mg. with Deserpidine 0.25 mg. or 0.5 mg

Indications: Edema and mild to moderate hypertension (Enduron), and mild to moderately severe hypertension (Enduronyl). More potent agents, if added, can be given at reduced dosage.

Contraindications: Sensitivity to thiazides; severe renal disease (except nephrosis) or shutdown; severe hepatic disease or impending hepatic coma (hepatic coma due to hypokalemia has been reported in patients on thiazides). Do not use Enduronyl in severe mental depression, suicidal tendencies, active peptic ulcer, or ulcerative colitis.

Warnings: Consider possible sensitivity where there is history of allergy or asthma. If added potassium is indicated, dietary supplementation is recommended. Reserve enteric-coated potassium tablets for cautious use only when necessary, as they may induce serious or fatal small bowel lesions (stenosis with or without ulceration), cause obstruction, hemorrhage, and perforation often requiring surgery; discontinue them immediately if abdominal pain, distention, nausea, vomiting, or g.i. bleeding occurs. Neither Enduron nor Enduronyl contains added potassium.

Precautions: Use thiazides cautiously in severe renal dysfunction, impaired hepatic function or progressive liver disease; also in pregnancy (bone marrow depression, thrombocytopenia, and altered carbohydrate metabolism have been reported in certain newborn). In surgery, thiazides may reduce response to vasopressors, and increase response to tubocurarine. Antihypertensive response may be enhanced following sympathectomy. Watch for electrolyte imbalance (e.g., hyponatremia) in all patients. In hypokalemia (especially in digitalized patients) give supplemental potassium. In hypochloremic alkalosis, give supplemental chloride.

Use rauwolfias with caution in patients with history of peptic ulcer. Rauwolfias with anesthetics may produce hypotension and bradycardia. Discontinue Enduronyl two weeks before elective surgery. Consider vagal blocking agents during emergency surgery. In epilepsy, adjust anticonvulsant dosage. In electroshock, shorten stimulus strength and duration. In occasional patients with depressive tendencies, rauwolfias may precipitate severe mental depression that usually disappears when drug is stopped.

Adverse Reactions: Thiazide reaction include blood dyscrasias (thrombocytopenia with purpura, agranulocytosis, aplastic anemia); elevation of BUN, serum uric acid or blood sugar; anorexia, nausea, vomiting, diarrhea, headache, dizziness, paresthesia, weakness, skin rash, photosensitivity, jaundice, symtomatic gout, and pancreatitis. Cutaneous vasculitis in the elderly has been reported with other thiazides. Adverse effects with deserpidine are qualitatively similar to those with reserpine, but their incidence is lower. These include nasal stuffiness, abdominal cramps or diarrhea, nausea, headache, weight gain, reduced libido and potency, peptic ulcer aggravation, epistaxis, skin eruption, asthma in susceptible patients, electrolyte imbalance, excessive salivation, and a reversible Parkinson's syndrome. Excessive drowsiness, fatigue, weakness, and nightmares may signal mental depression. Thrombocytopenia, purpura, and a symptom manifested by dull sensorium, deafness, uveitis, glaucoma, and optic atrophy are rare allergic reactions to other rauwolfias. Hypotension from antihypertensive agents may precipitate angina attacks in susceptible individuals. Usually adverse reactions disappear when drug is withdrawn.

EUTRON™

Each tablet contains Pargyline Hydrochloride 25 mg. with Methyclothiazide 5 mg.

Indications: For treatment of patients with moderate to severe hypertension, especially those with severe diastolic hypertension. Not recommended for patients with mild or labile hypertension amendable to therapy with sedatives and/or thiazide diuretics alone. It is desirable to establish the dosage of Eutron by administering component drugs separately.

Contraindications: Pheochromocytoma, advanced renal disease, increasing renal dysfunction, paranoid schizophrenia and hyperthyroidism. Hepatic coma has been reported as consequence of hypokalemia with thiazide therapy. Until further experience is gained not recommended for patients with malignant hypertension, children under 12, or pregnant patients.

Concomitant use of the following is contraindicated: other monoamine oxidase inhibitors; parenteral forms of reserpine or guanethidine; sympathomimetic drugs; foods high in tyramine such as cheese; imipramine and amitriptyline, or similar antidepressants; methyldopa. 2 week interval should separate therapy and use of these agents.

Methyclothiazide is contraindicated in patients with known sensitivity to thiazides.

Warnings: Pargyline hydrochloride is a monoamine oxidase inhibitor. Warn patients against eating cheese, and using alcohol, proprietary drugs or other medication without the knowledge of the physician. When indicated, alcohol, narcotics (meperidine should be avoided), antihistamines, barbiturates, chloral hydrate, and other hypnotics, sedatives, tranquilizers, or caffeine, may be used cautiously in reduced dosage. In emergency surgery ¼ to ⅕ the usual dose of narcotics, analgesics, and other premedications should be used avoiding parenteral administration where possible. Carefully adjust dose of anesthetics to response of patient. Withdraw pargyline two weeks before elective surgery.

Warn patients about the possibility of postural hypotension. Those with angina or coronary artery disease should not increase physical activity with an improvement in well being. Pargyline may lower blood sugar.

Avoid use of enteric-coated potassium tablets, as these may induce serious or fatal small-bowel lesions consisting of stenosis with or without ulceration. These small-bowel lesions have caused obstruction, hemorrhage and perforation frequently requiring surgery. Medication should be discontinued immediately if abdominal pain, distension, nausea, vomiting or GI bleeding occurs. These products contain no added potassium salts and if added potassium intake is desired, dietary supplementation is recommended. Coated potassium tablets should be reserved for cautious use when adequate dietary supplementation is impractical. In patients with a history of allergy or asthma the possibility of sensitivity reactions should be considered.

Precautions: Measure blood pressure while patient is standing to determine antihypertensive effect. Use with caution in hyperactive or hyperexcitable persons. Such persons may show increased restlessness and agitation. Withdraw drug during acute febrile illness. Watch patients with impaired renal function for increasing drug effects or elevation of BUN and other evidence of progressive renal failure; withdraw drug if such alterations persist and progress. Use with caution in patients with liver disease. As with all new drugs, complete blood counts, urinalyses, and liver function tests should be performed periodically. With prolonged therapy, examine patients for change in color perception, visual fields and fundi. Also reported have been: blood dyscrasias including thrombocytopenia with purpura, agranulocytosis and aplastic anemia; elevations of BUN, serum uric acid, or blood sugar. Symptomatic gout may be induced. In surgical patients thiazides may reduce response to vasopressors and increase response to tubocurarine.

Adverse Reactions: Pargyline may be associated with orthostatic hypotension. Mild constipation, slight edema, dry mouth, sweating, increased appetite, arthralgia, nausea and vomiting, headache, insomnia, difficulty in micturition, nightmares, impotence, delayed ejaculation, rash, and purpura have been encountered with pargyline. Hyperexcitability, increased neuromuscular activity (muscle twitching) and other extrapyramidal symptoms have been reported in a few patients with reduced cardiac reserve.

During intensive or prolonged therapy, guard against hypochloremic alkalosis and hypokalemia (especially the latter if patient is on digitalis). Observe all patients for signs of hyponatremia ("low salt" syndrome).

Reported thiazide reactions also include anorexia, nausea, vomiting, diarrhea, headache, dizziness, paresthesia, weakness, skin rash, photosensitivity, jaundice, and pancreatitis. Nocturia has been observed with the combination.

801438

ABBOTT

TM-TRADEMARK

Help can be weeks away
for a painful shoulder.

Except with Butazolidin® alka.

If it doesn't work in a week,
forget it.



lut don't forget this about Butazolidin alka

Contraindications: Edema; danger of cardiac decompensation; history or symptoms of peptic ulcer; renal, hepatic or cardiac damage; history of drug allergy; history of blood dyscrasia. The drug should not be given when the patient is senile or when other potent drugs are given concurrently. Large doses of Butazolidin alka are contraindicated in glaucoma.

Warning: If coumarin-type anticoagulants are given simultaneously, watch for excessive increase in prothrombin time. Instances of severe bleeding have occurred. Pyrazole compounds may potentiate the pharmacologic action of sulfonylurea, sulfonamide-type agents and insulin. Carefully observe patients receiving such therapy. Use with great caution the first trimester of pregnancy.

Precautions: Before prescribing, carefully select patients, avoiding those responsive to routine measures as well as contraindicated patients. Obtain a detailed history and a complete physical and laboratory examination, including a blood count. The patient should not exceed recommended dosage, should be closely supervised and should be warned to discontinue the drug and report immediately if fever, sore throat, or mouth lesions (symptoms of blood dyscrasia); sudden weight gain (water retention); skin reactions; black or tarry stools or other evidence of intestinal hemorrhage occur. Make regular blood counts. Discontinue the drug immediately and institute countermeasures if the white count changes significantly, granulocytes decrease, or immature forms appear. Use greater care in the elderly and in hypertensives.

Adverse Reactions: The most common are nausea, edema and drug rash. Swelling of the ankles or face may be minimized by withholding dietary salt, reduction in dosage or use of diuretics. In elderly patients and in those with hypertension the drug should be discontinued with the appearance of edema. The drug has been associated with peptic ulcer and may reactivate a latent peptic ulcer. The patient should be instructed to take doses immediately before or after meals or with milk to minimize gastric upset. Mild drug rashes frequently subside with reduction of dosage. However, rash accompanied by fever or other systemic reactions usually requires withholding medication. Purpuric rash has also been reported. Agranulocytosis, exfoliative dermatitis, Stevens-Johnson syndrome, or a generalized allergic reaction similar to serum sickness may occur and require permanent withdrawal of medication. Stomatitis, salivary gland enlargement, vomiting, vertigo and languor may occur. Leukemia and leukemoid reactions have been reported. While not definitely attributable to the drug, a causal relationship cannot be excluded. Thrombocytopenic purpura and aplastic anemia may occur. Confusional states, agitation, headache, blurred vision, optic neuritis and transient hearing loss have been reported, as have hyperglycemia, hepatitis, jaundice, and several cases of anuria and hematuria. With long-term use, reversible thyroid hyperplasia may occur infrequently. Moderate lowering of the red cell count due to hemodilution may occur.



Dosage in Painful Shoulder: Initial: 3 to 6 capsules daily in 3 or 4 equal doses. Trial period: one week. Maintenance dosage should not exceed 4 capsules daily; response is often achieved with 1 or 2 capsules daily.

6509-V(8)R2

Butazolidin® alka Geigy

Capsules

phenylbutazone, 100 mg.; dried aluminum hydroxide gel, 100 mg.; magnesium trisilicate, 150 mg.; homatropine methylbromide, 1.25 mg.

For complete details, please see full prescribing information.

Geigy Pharmaceuticals
Division of Geigy Chemical Corporation
Ardsley, New York 10502

BU 5462



Tissue's healing nicely. Yet anxiety slows his steps toward recovery.

By helping overcome anxiety and tension which can thwart the convalescent's progress, Equanil® often may play an important role in medical and surgical aftercare.

Indications: For use in management of anxiety and tension occurring alone or as accompanying symptom complex to medical and surgical disorders and procedures. Though not a hypnotic, fosters normal sleep through antianxiety and related muscle-relaxant properties.

Contraindications: History of sensitivity to meprobamate.

Important Precautions: Carefully supervise dose and amounts prescribed, especially for patients prone to overdose themselves. Excessive prolonged use has been reported to result in dependence or habituation in susceptible persons, as alcoholics, ex-addicts, and other severe psychoneurotics. After prolonged excessive dosage, reduce dosage gradually to avoid possibly severe withdrawal reactions. Abrupt discontinuance of excessive doses has sometimes resulted in epileptiform seizures.

Warn patients of possible reduced alcohol tolerance, with resultant slowing of reaction time and impairment of judgment and coordination.

Reduce dose if drowsiness, ataxia or visual disturbance occurs; if persistent, patients should not operate vehicles or dangerous machinery.

Side Effects include drowsiness, usually transient; if persistent and associated with ataxia, usually responds to dose reduction; occasionally concomitant CNS stimulants (amphetamine, mephentermine sulfate) are desirable. Allergic or idiosyncratic reactions are rare, but such reactions, sometimes severe, can develop in patients receiving only 1 to 4 doses who have had no previous contact with meprobamate. Previous history of allergy may or may not be related to incidence of reactions. Mild reactions are characterized by itchy urticarial or erythematous maculopapular rash, generalized or confined to groin. Acute nonthrombocytopenic purpura with cutaneous petechiae, ecchymoses, peripheral edema and fever have been reported. One fatal case of bullous dermatitis following intermittent use of meprobamate with prednisolone has been reported. If allergic reaction occurs, meprobamate should be stopped and not reinstituted. Severe reactions, observed very rarely, include angioneurotic edema, bronchial spasms, fever, fainting spells, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case) and hyperthermia. Treat symptomatically as with epinephrine, antihistamine and possibly hydrocortisone. Aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis and hemolytic anemia have occurred rarely, almost always in presence of known toxic agents. A few cases of leukopenia, usually transient, have been reported on continuous administration.

Meprobamate may sometimes precipitate grand mal attacks in patients susceptible to both grand and petit mal. Extremely large doses can produce rhythmic fast activity in the cortical pattern. Impairment of accommodation and visual acuity has been reported rarely. After excessive dosage for weeks or months, withdraw gradually (1 or 2 weeks) to avoid recurrence of pretreatment symptoms (insomnia, severe anxiety, anorexia). Abrupt discontinuance of excessive doses has sometimes resulted in vomiting, ataxia, tremors, muscle twitching and epileptiform seizures. Prescribe very cautiously and in small amounts for patients with suicidal tendencies. Suicidal attempts have resulted in coma, shock, vasomotor and respiratory collapse and anuria. Excessive doses have resulted in prompt sleep; reduction of blood pressure, pulse and respiratory rates to basal levels; and occasionally hyperventilation. Treat with immediate gastric lavage and appropriate symptomatic therapy. (CNS stimulants and pressor amines as indicated.) Doses above 2400 mg./day are not recommended.

Composition: Tablets, 200 mg. and 400 mg. meprobamate. Coated Tablets, WYSEALS® EQUANIL (meprobamate) 400 mg. (All tablets also available in REDIPAK® [strip pack], Wyeth.) Continuous-Release Capsules, EQUANIL L-A (meprobamate) 400 mg.

Wyeth Laboratories Philadelphia, Pa.

Wyeth® SERVICE TO MEDICINE

Equanil®
(meprobamate)
Wyeth

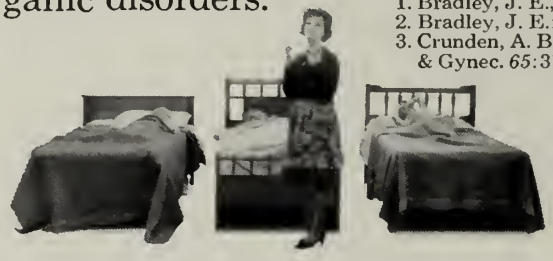
Professionally posed.



Togetherness....

...can be rough when epidemics of nausea and vomiting strike a family. Emetrol offers prompt, safe relief. It is free from toxicity[1] or side effects[2,3] and will not mask symptoms of serious organic disorders.

1. Bradley, J. E., *et al.*: J. Pediat. *38*:41 (Jan.) 1951.
2. Bradley, J. E.: Mod. Med. *20*:71 (Oct. 15) 1952.
3. Crunden, A. B., Jr., and Davis, W. A.: Am. J. Obst. & Gynec. *65*:311 (Feb.) 1953.



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Fort Washington, Pa.

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phosphorated carbohydrate solution
emesis control

To fight TB- find it first!

Make tuberculin testing routine with every physical examination.



TUBERCULIN, TINE TEST (Rosenthal)

Side effects are possible but rare: vesiculation, ulceration, or necrosis at test site. Contraindications: none, but use with caution in active tuberculosis. Available in 5's and 25's.

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DEPRESSION



NORPRAMIN®
(desipramine hydrochloride)

ANTIDEPRESSANT FOR RAPID IMPROVEMENT

At the recommended dosage level —initially, 150 mg. per day— gratifying remission of the signs and symptoms of depression typically begins in 2-5 days. Its specificity for depression, rapidity of action and usually mild side effects are significant reasons for prescribing NORPRAMIN (desipramine hydrochloride) in depression of any type . . . any degree of severity.

A few patients, sensitive to central nervous system stimulants may become restless as depression is lifted—in such cases dosage may be reduced or a tranquilizer added.

LAKESIDE

INDICATIONS: In depression of any kind—neurotic and psychotic.

CONTRAINDICATIONS: Glaucoma, urethral or ureteral spasm, recent myocardial infarction, severe coronary heart disease, epilepsy. Should not be given within two weeks of treatment with a monoamine oxidase inhibitor.

RELATIVE CONTRAINDICATIONS: (1) Patients with a history of paroxysmal tachycardia. (2) Patients receiving concomitant therapy with thyroid, anticholinergics or sympathomimetics may experience potentiation of effects of these drugs. (3) Safety in pregnancy has not been established. (4) Perform liver function studies in patients suspect of having hepatic disease.

PRECAUTIONS: (1) Outpatient use of desipramine hydrochloride should not be substituted for hospitalization when risk of suicide or homicide is considered grave. Suicidal ingestion of large doses may be fatal. (2) If serious adverse effects occur, reduce dosage or alter treatment. (3) In patients with manic-depressive illness a hypomanic state may be induced. (4) Discontinue drug as soon as possible prior to elective surgery.

ADVERSE EFFECTS: The following side effects have been encountered: dry mouth, constipation, dizziness, palpitation, delayed urination, agitation and stimulation ("jumpiness," "nervousness," "anxiety," "insomnia"), bad taste, sensory illusion, tinnitus, sweating, drowsiness, headache, hypotension (orthostatic), flushing, nausea, cramps, weakness, blurred vision and mydriasis, rash, tremor, allergy (general), altered liver function, ataxia and extrapyramidal signs, agranulocytosis.

Additional side effects more recently reported include: seizures, eosinophilia, confusional states with hallucinations, purpura, photosensitivity, galactorrhea, gynecomastia, and impotence. Side effects which could occur (analogy to related drugs) include weight gain, heartburn, anorexia, and hand and arm paresthesias.

DOSAGE: Optimal results are obtained at a dosage of 50 mg. t.i.d. (150 mg./day).

SUPPLIED: NORPRAMIN (desipramine hydrochloride) tablets of 25 mg.: bottles of 50, 500 and 1,000; and tablets of 50 mg. in bottles of 30, 250, and 1,000.

LAKESIDE LABORATORIES, INC., Milwaukee, Wisconsin 53201

What kind of douche?

With a plentiful supply of competitive products available, a Doctor has a real problem of selecting which of many similar products he should prescribe for his patients.

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For years PMC has been helping to maintain a balanced vaginal acidity favorable for protective Doderlein bacilli to thrive and grow. PMC produces and helps maintain a pH of 4.4. PMC is effective in reduction of Trichomonas vaginalis, monilia albicans, Staphylococci, Streptococci and Escherichia Coli. PMC is unexcelled in prescribing for vaginitis cervicitis, post-operative and post-partum use.

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Help clear up that miserable stuffed-up feeling with Dimetapp. Each hard-working Extentab brings welcome relief from the stuffiness, drip and congestion of upper respiratory conditions for up to 10-12 hours. Yet, patients seldom experience drowsiness or overstimulation. The key to success is the Dimetapp formula: Dimetane (brompheniramine maleate)—along with phenylephrine and phenylpropanolamine, two time-tested decongestants. They get the job done... in a hurry.

Indications: Dimetapp is indicated for symptomatic relief of the allergic manifestations of respiratory illnesses, such as the common cold and bronchial asthma. seasonal allergies. sinusitis, rhinitis, conjunctivitis, and otitis.
Contraindications: Hypersensitivity to antihistamines. Not recommended for use during pregnancy.
Precautions: Until patient's response has been determined, he should be cautioned against engaging in operations requiring alertness. Administer with care to patients with cardiac or peripheral vascular diseases or hypertension.
Side Effects: Hypersensitivity reactions including skin rashes, urticaria, hypotension and thrombocytopenia, have been reported on rare occasions. Drowsiness. lassitude. nausea, giddiness, dryness of the mouth, mydriasis, increased irritability or excitement may be encountered.
Dosage: 1 Extentab morning and evening.
Supplied: Bottles of 100 and 500.

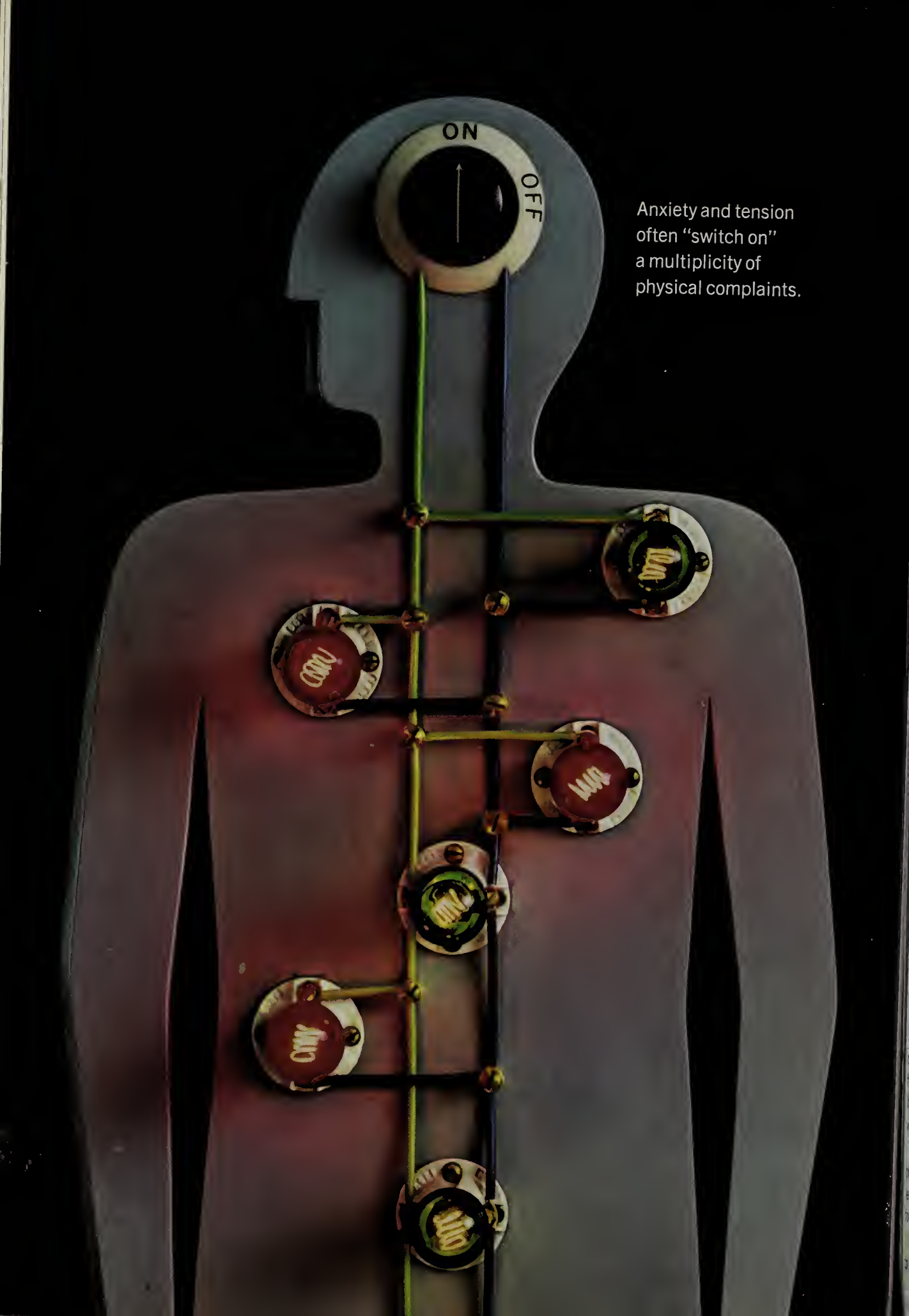
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RICHMOND, VA. 23220
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in sinusitis, colds, U.R.I.
Dimetapp® Extentabs®

(Dimetane® [brompheniramine maleate], 12 mg.; phenylephrine HCl, 15 mg.; phenylpropanolamine HCl, 15 mg.)

up to 10-12 hours clear breathing on one tablet





Anxiety and tension often "switch on" a multiplicity of physical complaints.

a tranquilizer with particular usefulness in functional disorders

Tybatran™

(pronounced TYE-buh-tran)

brand of tybamate

Extensive clinical experience, including eleven double-blind studies,[1-11] indicates that Tybatran is an effective agent for the relief of anxiety and tension. It appears to lend itself particularly well to the management of the anxious patient who "somatizes"—whose anxiety and tension find expression in complaints such as headaches,[4,8,10,11] fatigue,[4] insomnia,[2,4,8,9,12] anorexia,[3,8,9] and pruritus.[7]

Two salient features seem to set Tybatran somewhat apart from certain other commonly used tranquilizers.

1. Tybatran often proves more effective than meprobamate and chlordiazepoxide. In one study,[4] severe anxiety responded more effectively to tybamate than to meprobamate; in another,[8] symptom-response superiority of tybamate over chlordiazepoxide was marked at statistically significant levels of confidence.

2. Tybatran appears to be less sedating than other widely used tranquilizers. Side reactions are relatively infrequent; when they do occur, they may take the form of drowsiness, although insomnia, ataxia and other adverse effects have been reported. Nevertheless, Tybatran impresses many clinicians by its comparative lack of undesirable sedative action.[3,6,12,13] (If drowsiness or vertigo is present, activities requiring optimal alertness should be avoided.)

For patients in whom anxiety is manifested in any of a multiplicity of physical complaints, Tybatran deserves a clinical trial. These are the challenging patients, those with recurrent, persistent, ever-changing symptoms for which there is no clinical or laboratory evidence of organic disease.

Usual adult dose: one or two 250 mg. capsules 3 or 4 times daily. Adjust to suit individual requirements.

A·H·ROBINS

Prescribing Information

Dosage and administration. The suggested adult dose of Tybatran (tybamate) is one or two 250 mg. capsules three or four times daily. Dosage should be adjusted to suit individual requirements. While clinical experience with Tybatran (tybamate) in children has been very limited to date, the recommended daily dosage for children 6 to 12 years old is 20 to 35 mg./kg. body weight, in three or four equally divided doses. Dosage should be adjusted to suit individual requirements. Until further clinical experience is obtained, administration of Tybatran (tybamate) to children under 6 years of age is not recommended. Daily doses larger than 3000 mg. are not recommended, although in a few instances doses in excess of this figure have been administered. Tybatran (tybamate) is also available in 350 mg. capsules, for convenience in dosage adjustment, e.g., one capsule three times daily and two at bedtime.

Contraindications. Tybatran (tybamate) should not be administered to patients known to be hypersensitive to the drug. Since no studies have been done with this drug in human pregnancy, it should not be used in pregnancy unless the potential benefit outweighs the risk.

Warnings. Simultaneous administration to psychotic patients of tybamate with phenothiazines and other central nervous system depressants has in a few instances been associated with the occurrence of grand mal or petit mal seizures. Seizures have been reported with administration of phenothiazines alone, but not with administration of tybamate alone; nevertheless, tybamate should be used cautiously in individuals who are receiving other central nervous system depressants or have a history of convulsive seizures. Also, it should be borne in mind that simultaneous administration of tybamate with alcohol or with other psychotropic agents, particularly phenothiazines or monoamine oxidase inhibitors, which are known to potentiate the action of other drugs, may result in additive actions.

Precautions. There has been no evidence to date of the development of habituation or addiction. Investigators have not observed excessive self-medication or any withdrawal symptoms with use of Tybatran (tybamate), but the latter should be kept in mind with cessation of the drug after prolonged use. Because of the occurrence of

Prescribing information continued on next page.

(pronounced TYE-buh-tran)

Tybatran™

brand of tybamate

a tranquilizer with particular usefulness in functional disorders

Prescribing information continued from preceding page.

withdrawal symptoms or exacerbation of presenting symptoms upon rapid withdrawal of other agents of this type, abrupt withdrawal of Tybatran (tybamate) should be avoided. Tybamate, like other psychotherapeutic agents, should be used with caution in addiction-prone individuals. Should symptoms of hypersensitivity occur, administration should be discontinued at once and appropriate symptomatic treatment initiated. Operation of motor vehicles or machinery or other activities requiring optimal mental alertness should be avoided if drowsiness or vertigo is present. As with any new drug, Tybatran (tybamate), should be used with caution in patients with a history of drug allergies, blood dyscrasias, and hepatic or renal disease; and prolonged and/or high doses of tybamate should be accompanied by periodic measurements of hepatic, hematopoietic, and renal function.

Adverse reactions. While these have only rarely required discontinuation of the drug, the most frequently encountered reactions have included drowsiness, dizziness, nausea, insomnia, euphoria. The drug was discontinued in one child because of a possible drug-induced urticaria, and skin rash and pruritus have been encountered in a few other patients. In a few patients, effects suggesting excessive stimulation such as hyperactivity, fidgetiness, flushing, and tachycardia have been encountered. Other reported side effects recorded only a few times to date have included ataxia, unsteadiness, confusion, feeling of unreality, "panic reaction," fatigue, headache, paresthesias, vertigo, gastrointestinal disturbances, glossitis and dry mouth. Grand mal or petit mal seizures have been reported in a few hospitalized psychotic patients to whom tybamate (up to 6000 mg. daily), phenothiazines, and other psychotropic agents were administered simultaneously. Convulsive seizures have not been reported with the use of tybamate alone.

Until clinical experience has accumulated with this drug, inasmuch as tybamate is related to meprobamate, the physician should be cautious about the possibility of rare, serious adverse reactions such as may be encountered with the latter drug. Should excessive doses of tybamate be ingested, it is recommended that any drug remaining in the stomach be removed and symptomatic therapy, including central stimulants, be used as necessary.

Supply. Tybatran (tybamate) is available in green, sealed capsules of three strengths: 350 mg., 250 mg., and 125 mg. Each strength is supplied in bottles of 100 and 500.

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Guest Editorial . . . .

Medicine's Concealed Hemorrhage

I HAVE HAD NUMEROUS and at times pessimistic queries about what can be done concerning the financial predicament of hospitals. It is my opinion that a great deal could be achieved by physicians actively joining the board of trustees of their hospitals and participating in the deliberation of trustees. This could go a long way into curbing the financial and administrative excesses that exist today. To be more specific, I would like to enumerate some of the fields in which I feel these costly abuses can be corrected.

1. Salaries of Hospital Administrators—Mr. D. C. Carner, Administrator of Long Beach (California) Memorial Hospital has answered this for us. He states that "50% of hospital administrators earn between $32,500 and $37,500 a year—17% earn more." I feel this is excessive because there is not the challenge, the pre-eminence, nor the responsibility in hospital administration to justify such salaries.

2. Hospital administrators have created numerous positions at great expense to the hospitals. I have been amazed to find that hospital administrators have an assistant administrator, or administrators, director of personnel, and managers of various departments. All of these are highly salaried personnel and are performing duties which rightfully can be performed by one person, THE ADMINISTRATOR.

3. Other areas which could be delved into by physicians are fringe benefits, transportation, and the questionable necessity of all the meetings attended by hospital administrators. Numerous meetings are planned every year for the hospital administrators. These meetings entail a lot of traveling and last two or three days, at great expense to the hospitals. It has also been my experience that trivial matters are usually discussed at great length, and these meetings are usually unnecessarily prolonged, but the spirit of comraderie is wonderful to behold on the golf links and at the bars. The feeling of unmitigated joy comes from the con-

viction that we receive the best at no cost. On the other hand, utilization committees have been created and Medicare admissions have to be justified by signatures, all this in order to protect the public funds from medical extravagance. But then who is there to protect the same funds from administrative extravagance. Recently in our community it was reported that an official of our Community Hospital Association had spent, in less than a decade, in excess of one hundred seventy five thousand dollars ($175,000) for promotional purposes.

4. Then we have these institutes: the laundry, the housekeeper, the bookkeeper, central management, hospital labor relations, American Hospital Association of Pharmacy, the institute of hospital law, hospital purchasing, the institute of hospital personnel and more. In my opinion, all these institutes are not only costly in travel, entertainment, hotel expense and registration fees, but also these necessitate the absence of essential people from their work. I have found that one good institute would be mandatory to accomplish all this and that is the institute of "COMMON SENSE", with better supervision by the administrators.

5. Another area where expenditures could be trimmed is that of overstaffing in some departments. There is a sophistical attitude that stature is measured by the number of employees in the institution. I certainly feel that the employment of three people to change a light bulb is wasteful.

6. I feel certain hospital insurance plans are to blame for this proclivity for spending, the formula for reimbursement to hospitals is, by its very nature, conducive to extravagance, i.e., the greater the charges, the greater is the recovery of funds. It is therefore peremptory that this formula be changed for one in favor of economy; otherwise, financial chaos will drag the entire system of free medicine into a socialized one. It is my opinion that the best solution may be for Blue Cross and others to be made to operate like other insurance companies. Insurance carriers pay millions of dollars in taxes to their respective states (Blue Cross is tax exempt) and it may very well be that what appears to be a bargain through Blue Cross rates is in the long run just a FINANCIAL MIRAGE.

In conclusion, I believe that administrators have had a free hand in the field of hospital administration because they have been operating in what has been called a "no-man's land". The medical staff on one side and board of directors on the other, with a policy that the twain should never meet. The addition of medical men to the board of trustees (the number of trustees is flexible and can be changed at will, by the board) could bridge the gap and assist in decision that would enhance medical care and the efficiency of hospitals. It is certain that physicians as trustees can subject the hospital financial statement to microscopic scrutiny, and that way prune out wasteful expenditures.

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Sudden and Unexpected Deaths in Infancy

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An Analysis of 199 cases occurring in Virginia in 1965 with a discussion of etiological factors

SUDDEN AND UNEXPECTED DEATH in infancy, also known as "crib death", "cot death", or "sudden death syndrome", is a disorder characterized by the sudden death of an infant who had previously been well or affected with a mild illness such as coryza, cough, or a mild gastro-intestinal upset which was in no way felt to be of sufficient severity to have caused his death. Generally, it is agreed that the sudden death syndrome is a definite entity, the aetiology of which, be it single or multifactorial, is not known.

Every year in the United States alone, an estimated 25,000 infants die suddenly and unexpectedly. Representative figures reported in a survey by Valdes-Dapena[1] indicate 100 to 120 such deaths per year in Philadelphia, 100 in Cleveland and 400 in Los Angeles County; in Minneapolis, 10 percent of infant deaths fall in this category. In England and Wales, the reported figures vary between 1,090 and 1,400 infant deaths per year.[2,3] In 1952, 64 such cases were reported from Melbourne, Australia.[4] The sudden death syndrome is estimated to account for 20 percent of infant deaths in France.[1] In view of the seriousness of the problem of crib deaths and because no recent investigation of cases in Virginia has been made, it was decided to make a study of such deaths in this State. This paper analyzes 199 infant deaths reported to the Office of the Chief Medical Examiner of Virginia in the year of 1965 and compares and contrasts the features of the cases in Virginia with the findings reported in other studies. An attempt is made also to discuss the theories concerning the aetiology of this disorder.

From the Department of Legal Medicine, Medical College of Virginia, and Office of the Chief Medical Examiner, Commonwealth of Virginia.

Selection of the Cases

The criteria of sudden death syndrome accepted by us can be defined as: "The death of a child who was thought to be in good health or whose terminal illness appeared to be so mild that the possibility of a fatal outcome was not anticipated." [1] The deaths of infants and young children who had died of apparently natural, but not precisely determined, causes formed the bulk of the material. The cases that were clinically diagnosed as acute bronchopneumonia, gastro-enteritis, and aspiration of vomitus, but in which autopsy did not reveal any obvious cause of death also were included in the study. In addition, there were cases that initially seemed to fit the criteria of crib deaths, but in which autopsy revealed a definite cause of death. These cases were not excluded.

One of the responsibilities of the Office of the Chief Medical Examiner of Virginia is to investigate "unusual deaths" and deaths of persons who have not been attended by a physician during the course of their illness or at the time of death.[5] Although crib deaths may be classified as "unusual deaths", some physicians are aware of this entity as

a natural disease and do not report such cases for investigation. For this reason, it must be stressed that the cases studied here do not represent the entire number of crib deaths in Virginia in 1965. In all, 199 cases fitting the criteria of sudden death syndrome were selected for this study from a total of 217 cases reported to the Office of the Chief Medical Examiner of Virginia.

Analysis and Discussion of the Cases

The cases were analyzed with regard to age, sex, race, season, time of death, and geographical location. Other features like preceding symptoms, associated observations, and pathological findings are considered also. The results are discussed below.

Age: The distribution of the cases in different age groups is shown in the table below:

TABLE 1
SHOWING NUMBER OF DEATHS AT DIFFERENT AGES

Age in Months	< 1	1	2	3	4	5	6	7	8	> 8
Number of Deaths	22	41	39	36	19	8	6	7	8	13

Most of the cases fell in the age group of two weeks to eight months with the extremes being two days and three and one-half years. A great majority of deaths, 79.3 percent of the total, occurred in the first four months of life with a peak during the first month, and sustained during the second and third months. Three separate published reports reveal the peak age of infant deaths to be between two and four months,[6] three and six months,[2] and one and five months.[3] Generally, the consensus is that most of the deaths involve infants less than six months old.[1,4]

Sex: Generally, it is felt that there is a preponderance of males in the incidence of sudden unexpected infant deaths. Johnstone[3] reports 64 percent males, and other authors in a series reviewed by Valdes-Dapena note incidences of 55 percent to 83 percent of males. In that series, 25 out of 29 studies indicated a preponderance of males, while four studies noted either an equal percentage of males and females or slightly fewer males than females. Arey[7] and Cooke[8] do not think that the incidence is significant. In the present series, there were 105 (53 percent) males and 94 (47 percent) females. These figures show a slightly lower incidence of male involvement than do the majority of published reports. Of the 105 males, 57 were Negro and of the 94 females, 46 were Negro.

Race: Out of the 199 deaths in Virginia, 101 (50.7 percent) were Negroes. This figure indicates a disproportionate death rate since the population of Virginia is only 20 percent Negroes.[9] In the series reporting population studies, Negroes show 1.7 to 3 times greater incidence of sudden unexpected infant deaths than do Caucasians.[1] Gold[10] noted a rather high incidence of Negro deaths but gave no population percentage comparisons. Bowden[4] indicated that non-white deaths were four times as frequent as Caucasian deaths, and the figures in this series indicate the same ratio. Poor socio-economic conditions and standards of infant care may be the factors accounting for higher infant death rate in Negroes.

Geographic location: Over 53 percent of the population of Virginia lives in cities, suburbs, or small towns.[9] Of the cases analyzed here, 52 percent of the deaths occurred in urban areas, indicating no significant pattern of distribution of cases. Valdes-Dapena notes a number of reports, all from foreign countries, on the geographic distribution: Czechoslovakia and Austria reveal increased incidence in rural areas, Russia shows a high incidence at "the edges of the city", and Germany has an even distribution. In cities like Birmingham, England, and Philadelphia, U.S.A., the incidence is greater in depressed areas.[11,5]

Incidence by months of the year: There is general agreement throughout the literature that the majority of infant deaths occur in the colder months of fall, winter

and spring.[12] In Virginia, the pattern was the same; 64 percent of the deaths occurred in the six months of October through March. Even more striking is the fact that 38 percent of the deaths took place in the colder months of December, January, and February, whereas only 18 percent of the deaths occurred in the summer months of June, July, and August.

Time of day: In the present series, the distribution of deaths in the time period of 24 hours of day is shown in figure 1. This

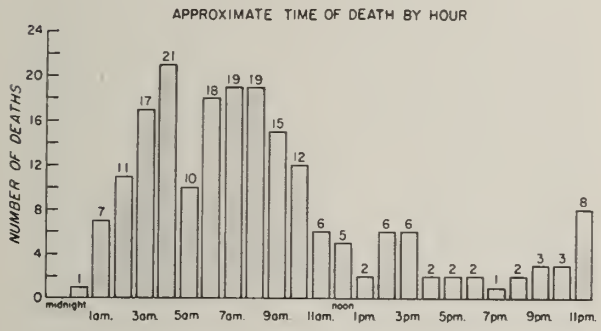


Fig. 1

points out that the majority of deaths occur in the early hours of the morning, 119 out of 199 deaths occurring between 3 a.m. and 9 a.m. This pattern of distribution is similar to that found by most investigators.[1,5]

Preceding symptoms: In our analysis, as far as anyone could tell, 88 (44 percent) infants were well and symptom-free before being found dead. About the same number had an antecedent respiratory infection which, in most cases, was reported as a "cold", "sniffles", and/or "cough". These symptoms generally were mild. A few infants had periods of dyspnoea but were not noted to appear critically ill. About 12 percent of the infants had suffered from some degree of gastrointestinal disturbance, vomiting being the most frequent and diarrhea rating next. Being "fussy" and "eating poorly" shortly before death was noted in three cases. These findings closely resemble those found by other workers in the field. A cold as a preceding symptom is frequent;[13,14] the present survey confirms this.

Other associated findings: Several factors seem to influence the incidence of crib deaths. Low socio-economic status,[11,15] poor infant care,[15] illegitimacy,[15] and prematurity[1] are associated with greater incidence of infant deaths. Other factors noted to influence the number of crib deaths are twin pregnancies,[1,8] young parents,[11] large families,[11,15] poor prenatal care,[1] and infant feeding with cow milk.[16] In the present study, poor economic level, poor care of infants, and large families were found to be the factors more significantly affecting the infant death rate. Only eight infants in the present series were noted to have been premature. Three of the 199 cases were one child of a set of the twins, and one was one child of a set of triplets. Two infants were mongoloid.

Pathological findings: In the present series of the 199 cases, postmortem examinations were performed on 57 cases. In nine cases, definite causes of death like bronchopneumonia (six cases), gastroenteritis (one case), fluoride poisoning (one case) were found. In the remaining 48 cases, the autopsy was in the conventional sense unrevealing. The usual findings were increased weight of the lungs, patchy or uniform purplish discoloration of surface feeling slightly firm, and congestion and oedema of the tissue. Microscopically, the lungs revealed congestion and patchy edema. The alveolar walls were thickened with an infiltrate of lymphocytes, occasional neutrophils and monocytes. In some areas, alveolar atelectasis could be seen and occasional alveoli were found to contain macrophages and alveolar lining cells. Cultures of lung tissue were either negative or revealed contaminants or non-pathogens.

Cultures of blood were always negative. In some cases, other findings like aspiration of vomitus, slight enlargement of thymus, and petechial hemorrhages on the thymus,

lungs, and the heart were seen. The published literature reveals that 75 percent to 85 percent of the autopsies show findings similar to those described above. In this study, approximately 84 percent of the postmortem examinations revealed the picture of the widely accepted diagnosis of interstitial pneumonitis.

Etiology of the Sudden and Unexpected Deaths in Infants

The etiology of crib deaths is still unknown despite a great deal of investigative work. There is a wide diversity of thought on the subject, and the thinking has changed with the times. A leading article in the British Medical Journal reveals: "Two or three generations ago doctors were satisfied with the explanation that cot deaths were due to 'teething', and in 1839 the Registrar-General's returns ascribed 5016 infant deaths to this cause." Several years ago, these deaths frequently were ascribed to *suffocation* by bed clothes, pillows, or overlying by a parent. In 1945, Woolley noted that over a five-year period in New York City, 139 infants were reported as smothered.[17] There is no doubt that the infants may die of suffocation, and, in fact, in 1965 the British Ministry of Health reported a significant correlation between the use of soft pillows and the incidence of sudden unexpected death in infants.[2] A careful investigation of the case, however, should be made before a diagnosis of suffocation is given, as, even today, some cases of sudden death syndrome are misdiagnosed as suffocation.

Another cause of sudden death popularly accepted a generation ago was "*status thymolymphaticus*" which was supposed to be responsible for asphyxia by "tracheal and auricular compression and distortion."[18] This cause generally is discarded by modern investigators; and as an expression of uniformity of thinking, The Lancet, one of England's most critical medical journals pronounced[19] "without sorrow, the end of the status" in 1931.

In recent years, several theories have been advanced to explain the cause of infant death. The majority of the workers agree that the death is from *interstitial pneumonitis*, an inflammation of lung tissue for which no cause is known. This diagnosis is based on the findings in the lungs. In rebuttal of this diagnosis, however, Valdes-Dapena[5] states: "Most pathologists who have had significant experience with small infants . . . now recognize that the so-called thickening of inter-alveolar septa which has been erroneously interpreted as interstitial pneumonia is no more than is normal in infants of this age, whose alveolar septa are routinely somewhat thicker and more cellular than those of an adult."

Bacterial infection has been incriminated by some as a possible cause of the condition because of frequent association of respiratory symptoms and seasonal incidence paralleling that of lung infections. The most striking results were published by Johnstone and Lawy.[3] They reported the presence of lower respiratory tract pathogens in 37 of the 55 carefully studied cases. Their review of literature indicates isolation of pathogens in from 7 to 58 percent of the cases. Cooke and Welch[8] isolated organisms in over 5 percent of their cases, but, in general, described bacteriological studies as "unrewarding". It seems likely that fulminating bacterial infection may be responsible for some of the crib deaths.

The possibility of *viral infection* as causing the changes in the lungs has been recognized widely. The results of careful search for the entero-viruses and respiratory viruses have, however, been mixed as is clear from the table on following page.

In addition, Johnstone and Lawy[3] and Cameron and Asher[11] report negative virus studies in their series. The reason for the wide divergence of the positive virus isolations is not known. The works do, however, point to the importance of viral

studies, and it is conceivable that the work in this direction will be more fruitful as viral culture techniques improve.

Author	Total Cases	Positive Isolations	Virus Found
Gold[10]	48	12	Enterovirus, esp. Coxsackie A
Moore et al.[21]	10	7	Echo 7 in most cases. 1 each of Polio 1 and 2
Valdes-Dapena and Hummeler[20]	109	1	Coxsackie B
Horstmann and Hsiung[22]	3	3	Para-influenza, type 2
Balduzzik-Greendyke [13]	97	14	Coxsackie B
Parish et al.[23]	8	0	No isolation

Hypersensitivity to cow's milk antigens as a possible cause of crib deaths was proposed by Parish et al.[24] in 1960. They suggested that sudden death might be caused by anaphylactic reaction after aspiration of cow's milk by an infant who has become sensitized to proteins contained in such milk. These authors confirmed their findings of increased levels of milk antibodies in cases of sudden infant deaths in their second article[23] published in 1964. Perhaps on the basis of these papers, the Ministry of Health in Britain reported a statistically significant relationship between cot deaths and artificial feeding. On the other hand, however, Peterson and Good[25] noted that chronically ill and debilitated children have higher levels of precipitins and hemagglutinating antibodies than well or acutely ill children. Johnstone and Lawy[3] found no difference between the serum milk antibody titres in cases of cot deaths and in normal live infants. Gold and Adelson[16] came to the same conclusions and indicated that there was no evidence to support the hypothesis that hypersensitivity to milk was causally related to the sudden death syndrome.

Sudden death in infants or adults may be entirely *neurogenic* (Adelson, 1953).[26] Strong vagal impulses may cause cardiac arrest and lead to fatal cerebral anoxia. Adelson summarized his theory of neurogenic death by stating, "The essential factor in death from inhibition is the instantaneous establishment of an inhibitory reflex with the sudden arrest of a vital organ by what has been termed 'micro-trauma', injury of so minor a degree as to leave no or minimal visible evidence at autopsy." Stowens[27] believes that many stimuli, frequently "microscopic in extent" could produce fatal viscero-visceral reflexes. Another version of the neurogenic theory has been described by Wolf.[28] He states, " . . . one can reasonably suspect that a host of bizarre sudden deaths from 'fright', unexplained deaths in swimming pools and 'crib' deaths may be attributable to an over-exuberant oxygen conserving reflex."

Hypogammaglobulinemia was suggested by Spain, Bradess, and Greenblat[29] as an etiology of crib deaths because they found decreased levels of gamma globulin in three cases of sudden death syndrome. Valdes-Dapena et al.,[30] however, tested sera of 114 sudden deaths and 33 controls and found no significant difference.

Gunther[31] considered the possibility of infant death from *anaphylactic shock* following intra-uterine infection. He cited the example of rubella infection acquired in utero and noted that the virus could be isolated from such an infant many months after birth, while in a postnatal infection it was no longer isolated only a few days after the illness. In addition, infants with intra-uterine infection with rubella have been shown to have a high antibody titer

while the virus is still in the body. Gunther, therefore, felt that in this unusual situation an antigen-antibody reaction might take place with resultant anaphylactic shock and death. The fact that so many infants die in winter months and between two and six months of age has been explained by the ideas that the initial infection was acquired by the mother in the first trimester a year earlier during the winter when the incidence of viral infection is higher and that the immunologic state becomes ripe for the fatal reaction when the infant is two to six months old.

Reiders et al.[32] reported in 1964 the finding of the formation of a *"red substance"* when using the Stas-Otto procedure for deproteinization of brain tissue in infants dying from the sudden death syndrome. This red material, in the final alcoholic filtrate, was felt to be a very polar material, "probably phenolic" and not a protein. Of the cases in which the red material was found, 86 percent were crib deaths, while the cases in which the material was absent, only 23 percent were crib deaths. The significance of this material in crib deaths is unknown.

Some workers have considered *metabolic disorders* as possible causes of crib deaths. Porter[33] proposed the theory that factors like the immaturity of the neonatal liver, the presence of a subclinical amino acid metabolic disorder, and the overloading of the bottle fed infant with protein combine to precipitate a metabolic crisis. According to Stowens et al.,[34] the crib deaths are the result of an anaphylactic reaction to "a protein" absorbed from the gastro-intestinal tract. They go on to explain that the absorption of the antigen causes the infants' immune reactions to be maximally active which accounts for the hypertrophy of the thymus, the organ "responsible for the initiation of such mechanisms". The cause of death is thought to be an anaphylactic reaction that occurs when natural loss of neutralizing antibodies passively acquired from the mother reaches a critical level. These theories have not, however, been confirmed.

Congenital abnormality, possibly acquired in early foetal life, has been suggested by Geertinger[35] as a possible cause of crib deaths. Based on the histological study of serial sections of the thyroid region in 122 consecutive cases of sudden, unexpected death in the first year of life, he suggested that crib deaths are correlated with an incomplete embryological development of the parathyroids and that the cause of this poor development of parathyroids is probably in the calcium metabolism of the mother early in pregnancy. Valdes-Dapena[1] disputes Geertinger's theory by pointing out the unlikelihood of absent parathyroids in the presence of normal thymus as both of these are derived from the same embryologic structure. She also adds that the infants with congenital absence of the parathyroids usually die at about two weeks of age after overt tetany and not at two months of age with an antecedent history of good health.

General Discussion and Summary

One hundred and ninety-nine cases of sudden unexpected death in infants occurring in Virginia in the year 1965 are analyzed and the findings are compared with those in other studies reported in the literature. It is clear that there is general agreement with the findings of others in that these cases tended to involve infants of less than six months of age, they were more frequent in the colder months, death tended to occur in the early morning hours, and there was frequently a mild preceding illness not felt to be of a life threatening nature. It is obvious also that the infant death rate is significantly higher in Negroes. This may be due to differences in the economic status and standards of infant care. The majority of the infants showed symptoms of a minor cold or no symptoms at all prior to death.

A short summary of the etiologies of the sudden death syndrome is presented. It is apparent that there is no general agreement

about the cause of this condition. Almost every theory suggested concerning the pathogenetic mechanism of crib deaths has been shown to be unsatisfactory. It seems most likely, however, that the deaths are the results of viral infection; and research in the direction of isolating viruses from all living infants suffering from colds, in addition to a study of contacts and victims would be most profitable. Another important factor probably is anoxia, as can be seen from the anoxial changes in the organs at autopsy. A combination of factors like viral infection, poorly ventilated rooms, tight bed-clothes, and soft pillows may lead to anoxia.

It appears that the awareness of this condition is less than adequate among the public and even the physicians. The awareness among physicians has been improving as can be seen from the increasing number of cases reported to the Office of the Chief Medical Examiner in Richmond, over the period of six years:

Year	*Cases Reported*
1961	38
1962	51
1963	110
1964	195
1965	217
1966 through June	131

Several organizations, like the Washington State Association for the Study of Sudden Infant Death, The California Association for Sudden Infant Death Study, The Guild for Infant Survival, The Mark Addison Roe Foundation in Greenwich, Connecticut, and the National Foundation for Sudden Death in New York City, are operating to increase the understanding of the entity. Such an organization in Virginia would serve a very useful purpose. The deaths are completely unexpected and bring sudden shock and feelings of guilt to parents. In order to prevent the guilt reactions in the parents of victims, public understanding of the sudden death syndrome should be improved. Every physician must stress that this leading cause of death in infants is a disease probably caused by a virus; that it is not caused by suffocation; that it is not possible to detect it during life or prevent it, as it is not associated with any specific signs or symptoms; that it is often symptom free and that death is not the result of an act of omission or commission.

In view of the lack of knowledge of exact cause of crib deaths, no specific preventive measures can be advanced. The general advice to parents with possible preventive significance should include care of the infant in the first year of life along the following lines:

1. Feed the infant in a sitting position. Do not leave the infant in the crib with the bottle of milk. If possible, breast feed during the first six months of life.
2. Dress the infant in loose clothes for sleeping, and do not use soft pillows.
3. Ventilate the infant's room intermittently.
4. Consult a physician as soon as an infant develops a cold or respiratory symptoms.

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Criteria for Diagnosis of Asthma, Chronic Bronchitis and Emphysema

With a Note on Pink Puffers and Blue Bloaters

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The patient with chronic bronchitis may wheeze. Does he also have asthma or emphysema? This is an attempt to define these diagnoses precisely.

IN MAY OF 1962 the Committee on Diagnostic Standards for Non-Tuberculous Respiratory Diseases of the American Thoracic Society[1] set forth definitions and classifications for "chronic bronchitis", "asthma", and "emphysema". As stated, this was to provide a trial period in which clinicians, pulmonary physiologists, pathologists and other interested persons might "put them to the test of daily usage and practice". Since that time we have tried to utilize these terms in the daily diagnosis, management and counseling of patients with "chronic lung disease". The following comments are made in the light of this clinical experience and include a brief discussion of the descriptive clinical terms "Blue Bloater and Pink Puffer".

The need for criteria for differentiation of these clinical entities has become increasingly evident since experience has shown that each, when properly defined, differs in pathophysiology, effective treatment, and prognosis. As the categorization of "chronic lungers" proceeds, certain facts have become apparent which were previously unappreciated, i.e., asthma does not lead to emphysema, chronic bronchitis is a common American as well as English disease, and Cor Pulmonale may be due to hypoxemia without parenchymal lung disease, so that chronic bronchitis can kill without associated emphysema.

Definitions

A review of the "Definitions and Classification of Chronic Bronchitis, Asthma and Pulmonary Emphysema" may be summarized as follows:

Chronic Bronchitis. Chronic bronchitis is a clinical disorder characterized by cough with production of sputum. Arbitrarily cough and sputum must be present on "most days for a minimum of three months in the year and for not less than two successive years". Obviously this is a diagnosis of exclusion since a number of pulmonary diseases may have identical symptoms. Classically a patient with chronic bronchitis is a male, older than 35 years of age who smokes cigarettes, coughs and spits each morning, and may have recurrent episodes of fever, purulent sputum and wheezing. Pulmonary function tests show primarily increased resistance to air flow. Chest x-rays may be normal unless the disease is complicated by pneumonia. Many patients with emphysema have histories of long standing chronic bronchitis. The relationship between these two entities is discussed below in some detail.

Asthma. Asthma is a disease characterized by an increased responsiveness of the trachea and bronchi to various stimuli manifested by a widespread narrowing of the airways

that changes in severity either spontaneously or as a result of therapy. These stimuli, which result in periodic excessive contraction of smooth muscle and hypersecretion of mucous in the bronchial tree, may be of allergic origin, may be due to acute respiratory infection or emotional reactions. In many individuals the inciting stimulus cannot be discovered. The term "asthma" should not be used for the bronchial narrowing which occurs in chronic bronchitis or from obstructive diseases of the lung such as pulmonary emphysema. As one of my mentors is fond of saying, "asthma is not protective" so that chronic bronchitis, emphysema and asthma may occur in the same individual, but here the airway obstruction of asthma is not causally related to these diseases. Obstruction to airflow in asthma is present during expiration and inspiration, so that wheezes may be heard during both phases of respiration.

Emphysema. Emphysema is an anatomic alteration of the lung characterized by an abnormal enlargement of the air spaces distal to the terminal non-respiratory bronchiole accompanied by destructive changes of the alveolar walls. The degree of the severity of the clinical manifestations is generally related to the percentage involvement of the lungs in the destructive process. The thoracic cage is expanded with limited motion. There is visible prolongation of the expiratory phase of respiration. There is generalized hyperresonance to percussion with suppression of breath sounds and there may be rhonchi and wheezes. Except when bullae are present the chest x-ray may show only hyperinflation, and cannot be distinguished from asthma or over-inflation of the lungs due to other causes. Pulmonary function tests show a reduction in the rate of expiratory air flow (reduction in timed vital capacity and maximum mid expiratory flow rate which may result in a reduction in the maximum voluntary ventilation). Vital capacity may be increased, normal, or reduced. Classically, symptoms are brought on by exercise and abate with rest.

Comment

Chronic Bronchitis. The major recent change in the concept of chronic bronchitis has been the increasing evidence that it may frequently occur in its most severe form as an entity separate from emphysema. For some years the British workers have been aware that patients with progressive chronic bronchitis may have obstructive airway disease, hypoxemia, Cor Pulmonale, and die, and at autopsy have no evidence of significant emphysema. We have been slow to accept this concept in the United States. However, documented cases have been reported in recent years.[2,3] In a patient with chronic bronchitis and physiologic evidence of airway obstruction one is therefore on more firm scientific ground with the diagnosis of "chronic bronchitis and obstructive pulmonary disease", rather than the diagnosis of "chronic bronchitis and pulmonary emphysema".

Asthma. This more precise definition of "asthma" provides a distinct and useful diagnostic category and should survive as pertaining to a specific clinical entity and not merely to the non-specific physical finding of wheezing. The description of "asthma" as a disease characterized by an "increased *responsiveness* of the trachea to *various* stimuli" appears more realistic than the artificial limitation of the term to manifestations of allergic disease. It becomes extremely important from a prognostic point of view to correctly diagnose "asthma" since "emphysema" appears to be a rare complication of this condition.[3,4,5]

Emphysema. The strong point in the definition of "pulmonary emphysema" is at once its weakness—a classification based on pathologic findings. Certainly a more rational basis for definition is provided by morphologic rather than clinical criteria. However, this does not give immediate aid

to the practitioner dealing with living patients. A certain percentage of the lung must be involved before clinical findings can be attributed to this condition. For this reason proper inflation prior to fixation for both gross and microscopic examination is necessary. The pathologic differentiations of Panlobular and Centrilobular emphysema promised to afford an opportunity for correlations between morphologic abnormalities and function. However, this has not been the case as discussed below.

Pink Puffers and Blue Bloaters

It is usually possible to separate most patients with severe obstructive pulmonary disease into two clinical categories—the Pink Puffers and the Blue Bloaters. According to Scadding[6] the terms "Blue Bloater" and "Pink Puffer" originated with Dr. A. C. Dornhorst of St. George's Hospital in London. To quote Dr. Scadding, "The Blue Bloaters are most characteristically those who have started with long continued chronic bronchitis punctuated by recurrent inflammatory episodes. After many years these episodes are accompanied by alveolar hypoventilation and cyanosis, carbon dioxide retention with associated disorders of consciousness often made worse by oxygen, and edema and raised jugular venous pressure. With appropriate treatment they may survive several of the episodes of edema to which the term 'Bloater' refers. In such patients emphysema may not be a prominent feature at necropsy although during life severe airway obstruction is found.

"The Pink Puffer, considerably less frequent in England, is the type of patient who starts with progressive dyspnea on exertion, usually in middle age, without a preceding history of chronic bronchitis. He has overdistended lungs, often curiously silent to auscultation. In spite of a much reduced ventilatory capacity with considerable irreversible airway obstruction and greatly increased residual capacity, he nevertheless manages to maintain a minute volume above that of a normal subject at rest and so keeps his PCO_2 down to normal levels until very shortly before the end of his disease. Moreover, he develops right ventricular failure, if at all, only terminally, very rarely making a useful functional recovery once edema has appeared. Such patients may confidently be expected to show emphysema at necropsy."

There has been recent confirmation of Dr. Scadding's suggestion that there is indeed a correlation between clinical types and pathologic findings. In a combined study,[7] English and American patients with airway obstruction could be divided into two clinical categories. In Type A patients with "emphysematous airway obstruction" (Pink Puffers), the diffusion capacity was much impaired as measured by the single breath CO technique. These patients pathologically showed extensive emphysema (the morphologic type of emphysema did not have any special significance). The Type B patient with "bronchitic airway obstruction" (Blue Bloaters) had a normal diffusion capacity and showed minimal if any emphysema at autopsy. Unfortunately there was also a third group of patients in whom clinical criteria were not helpful, but these comprised less than one quarter of the total studied.

This is as much as can confidently be said about these two categories of patients at present, except that the Blue Bloater tends to be stocky, may not be overtly dyspneic and has "chronic bronchitis"—a non-fighter. The Pink Puffer may have had significant weight loss, usually has minimal cough, may be pitiously short of breath, and fights for life throughout. This difference in the response of patients to an increased respiratory work load has been discussed editorially by Robin[8] who feels that the fundamental basis for this difference may be neurogenic, although specific evidence for this is lacking.

Summary

"Chronic bronchitis" is a historical diagnosis made in patients who may become in

time "Blue Bloaters". "Emphysema" is primarily a pathologic diagnosis with increasing evidence that it has a correlation with the clinical syndrome characterized by "Pink Puffer". The term "Asthma" is used to define a disorder of pulmonary function due to a variety of etiologic agents that can be detected by physiological tests and usually suspected by history.

To again quote Dr. Scadding "The more precisely words are defined the more efficient they are as a means of expressing our knowledge and the less use as a cloak for ignorance."

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Clinical Center Studies of Bilirubin Metabolism

The cooperation of physicians is requested in the referral of patients with hyperbilirubinemia for studies of bilirubin metabolism being conducted by the Metabolism Branch of the National Cancer Institute at the Clinical Center, National Institutes of Health, Bethesda, Maryland.

Of particular interest are patients with chronic or intermittent unconjugated hyperbilirubinemia, in whom the diagnosis of hemolysis, Gilbert's syndrome, or a post-hepatitic syndrome is suggested. Patients with conjugated or mixed hyperbilirubinemia (e.g., Rotor's syndrome or Dubin-Johnson syndrome) in whom obvious obstructive or active inflammatory disease has been ruled out, are also needed. A few patients with stable, compensated cirrhosis of the liver may also be accepted for study.

Selected patients will be admitted to the Clinical Center as inpatients for a minimum period of two weeks. Isotopic bilirubin clearance and red blood cell life-span studies, as well as dye clearance studies, will be performed. Liver biopsy may be performed in selected situations. Upon completion of their studies, patients will be returned to the care of the referring physician who will receive a summary of findings.

Physicians interested in having their patients considered for admission to these studies may write or telephone: Paul D. Berk, M.D., Clinical Center, Room 4-N-117, National Institutes of Health, Bethesda, Maryland 20014, Telephone: 656-4000, Ext. 65955 (Area Code 301)

Removal of Ear Wax

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The removal of an undesirable accumulation of wax from the ear should be done with care and with a knowledge of previous perforation or infection. This is a task for the physician and not for the office assistant.

WAX ACCUMULATION is a frequent finding by physicians and a rather common complaint of patients. The regular occurrence of the problem and the potential difficulties that may be encountered makes this a timely topic warranting review. Cerumen is the normal secretion of the cerumenous glands. These glands are located in the subcutaneous tissue of the cartilagenous portion of the external auditory canal. It is continually formed in such small quantities that its presence is usually unnoticed. Wax is discharged regularly from the auditory meatus. The action of the mandible in mastication and speaking causes a motion of the anterior and inferior walls of the auditory canal. This results in a gradual movement of wax externally. During its presence in the auditory canal cerumen functions as a lubricant to prevent absorption of water into the skin. During the progress of migration externally exfoliated skin of the canal is removed with the wax.

Symptoms

When wax completely occludes the canal or comes in contact with the tympanic membrane symptoms will occur. They vary depending upon the consistency of the wax, size of the accumulation, and location of the mass. It is not unusual during a routine physical examination to find a large accumulation of which the patient is unaware. If there is a small opening between the canal wall and the wax there will probably be no noticeable loss of hearing. Occasionally, when a thin layer of soft wax bridges across the canal a recurrent mild hearing loss will occur. Digital pressure by the patient manipulating the ear canal will often temporarily relieve the blockage. If the tympanic membrane is suddenly covered with wax or skin debris from the canal there will be a pronounced hearing loss and fullness in the ear. Tinnitus and vertigo may occur. Autophony, an increase in the sound of the patients own voice in the affected ear, is often an additional complaint. Pain is relatively rare.

Causes of Wax Accumulation

Any process interfering with the regular movement of the wax will lead to an accumulation. The following are the most frequent causes:

1. A small auditory canal, especially in children.
2. Abnormal contour of the canal.
3. Excessive amount of wax formation, more frequent in people with an oily skin.
4. Large quantities of thick hair in the canal. This mechanically collects the wax to form a mass, especially in older men.
5. Hypersecretion of the cerumenous glands due to irritation of the skin, often due to self induced trauma by the patients use of metallic implements.

6. Moisture forming in or entering the ear. Perspiration can loosen the mass. Improper methods of washing may accidentally allow water or soapsuds to enter. The first swim of the year will often move an accumulated mass inward against the drum membrane.

Past History

Obtaining an accurate history of prior ear difficulty is very important. Previous earache, discharge, and hearing loss would indicate that a perforation had existed. If discharge reoccurred or hearing loss persisted the possibility of a perforation of the tympanic membrane is very likely. Occasionally, a previous perforation will have healed with a very thin atrophic scar. Such a thin membrane is susceptible to perforation again if a forceful stream of water strikes its surface during irrigation.

Diagnosis

Inspection of the ear canal will demonstrate the extent and location of the wax accumulation. The use of a head mirror will sharply focus the light into the ear. Both hands are then free for manipulation of instruments. Not infrequently the canal may be adequately evaluated without introducing an ear speculum. Early in its formation wax is very thin with a light yellow color. Later it becomes thicker and darker due to exposure to air. The following may be mistaken for cerumenous plugs:

1. Purulent secretion covered by skin debris and a layer of wax.
2. Hard dry crusts lining the canal; the dried aftermath of a previous accumulation of purulent discharge.
3. Small foreign bodies enveloped in cerumen. A small ball of cotton might be lost by the patient and forgotten. Children can introduce an object into the ear that may remain without any symptoms.

Removal of ear wax should never be delegated to the physician's assistant. The potentiality of mild to severe trauma with pain, bleeding, or even perforation of the tympanic membrane is always present. Otitis externa, due to a laceration of the thin skin, may occur. If water from the irrigation is not dried thoroughly it may be absorbed by the skin. This results in maceration of the tissues and diffuse inflammation. If a previous perforation exists, or a traumatic perforation occurs, water from the irrigation will enter the middle ear through the perforation. Purulent otitis media may then develop.

If small accumulations or a dry mass are present near the external opening, I prefer to avoid irrigation. A smooth curette or small forceps (Fig. 1) will usually allow the

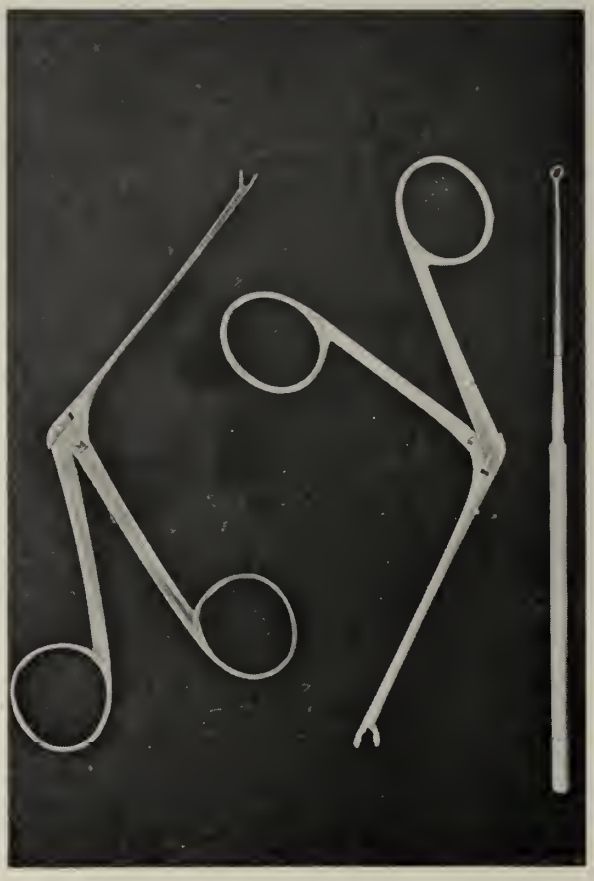


Fig. 1.

wax to be removed satisfactorily. If the mass is rather soft or located medially near the drum surface, irrigation is necessary.

Comfortably warm water in a syringe is used. The stream of water is directed posteriorly and superiorly to allow the pressure to pass between the mass and the posterior canal wall. The water then strikes the surface of the tympanic membrane and returns along the anterior wall to the medial surface of the wax. This forces the mass out of the canal. If the current of water impinges

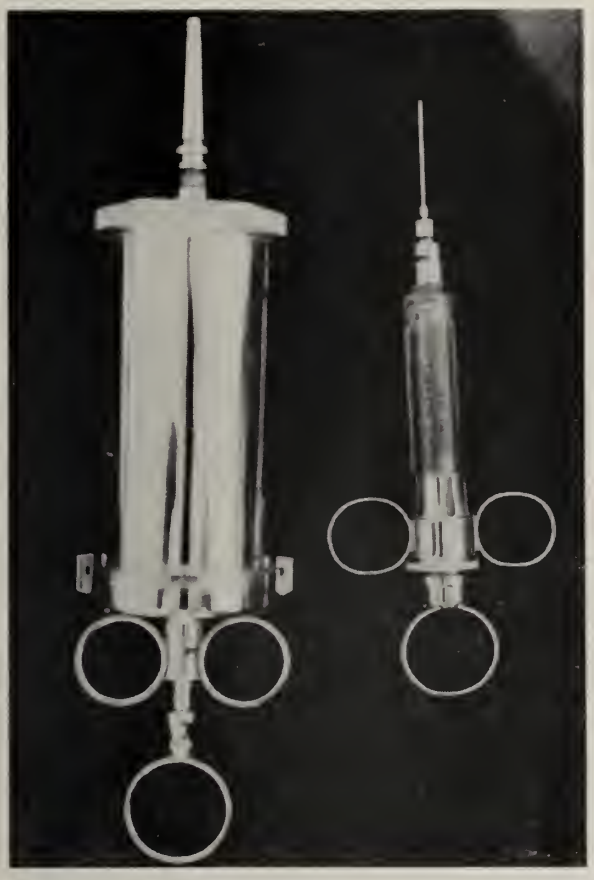


Fig. 2.

directly on the center of the lateral surface of the mass it will be forced inward.

The large metal ear syringe (Fig. 2) is used routinely by many physicians. The size varies from two to six ounces, making it awkward to hold in the hand. Considerable force is usually required to depress the plunger. Visualization of the canal is difficult, especially when the olive shaped tip is attached. The force of the stream of water can create immense pressure against the drum membrane.

I prefer to use a 5cc Luer-Lok syringe with metal thumb and finger rings. (Fig. 2) An 18 gauge needle is attached. The point has been removed by filing. This syringe and needle is small enough to allow good visualization of the ear canal. The thin stream of water has sufficient force to deliver the mass to the external meatus. Very little chance of trauma is possible.

Following the procedure the entire canal and surface of the drum membrane must be dried gently with a small piece of dry cotton wrapped tightly on the end of a metal applicator. This should reduce the possibility of the skin infection later.

Summary

The formation, accumulation, and removal of ear wax is reviewed. The small syringe for irrigation is described and discussed. I hope it will be useful to other physicians.

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Current Considerations in the Management of Adenocarcinoma of the Colon and Rectum

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Earlier diagnosis and treatment is of primary importance in improving the outlook for cases of carcinoma of the colon and rectum.

AN ESTIMATED 73,000 new cases of adenocarcinoma of the colon and rectum will be diagnosed in 1967. Unfortunately, approximately 44,000 patients will die from this disease the same year.[10] These figures are a grave reminder of the magnitude of the problem and the need for improved treatment. This paper presents current considerations in the management of these patients.

Diagnosis

The best diagnostic tools are still a complete history and physical examination. Change in bowel habits, bleeding, anemia, pain, weight loss and intestinal obstruction should immediately alert the physician to the possibility of a colonic malignancy. Digital examination of the rectum will diagnose 20-30 percent of the carcinomas of the colon and rectum. Seventy-five percent are within the range of the sigmoidoscope.

The barium enema x-ray examination is of great diagnostic help in locating lesions above the reach of the sigmoidoscope. Not only are the carcinomas located, but commonly associated lesions such as polyps, are found. More sophisticated studies, such as the silicone foam enema[15] and exfoliative cytology of colonic and rectal irrigation returns,[36] are currently being investigated without widespread practical application as yet.

Differential diagnosis is usually not difficult although the problem of sigmoid diverticulitis can be worrisome and the two conditions may co-exist.[43] Whenever there is doubt, early surgical excision is mandatory.

It cannot be emphasized too strongly that every physical examination should include a rectal examination. In person over 40, a sigmoidoscopy should also be performed. Any patient with persistent colonic symptoms should have, in addition to a thorough physical examination including digital rectal examination, a sigmoidoscopy and a barium enema.

Colorectal carcinoma is usually curable when the tumor is discovered and properly treated prior to its invasion of the muscularis mucosa of the bowel. Only when the physician maintains a high index of suspicion and performs the necessary diagnostic procedures will these lesions be found.[30,31]

Polyps

The close relationship of polyps to adenocarcinoma of the colon and rectum has definitely been established. Whether or not polyps are premalignant lesions is unknown. The important fact is that one cannot differentiate between a benign polyp and a carcinoma until it is removed and studied

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histologically. The only characteristic of small tumors beyond the range of the sigmoidoscope that correlates with the presence of cancer is size. Spratt and Ackerman[52] found only one carcinoma in 91 tumors less than 1.2 cm in diameter, while in 265 tumors larger than 1.2 cm, there were 227 carcinomas (85.7%). The high incidence of malignancy in papillary (villous) adenomas, 33 percent, as opposed to adenomatous polyps, 2 to 3 percent, has been emphasized.[35] Familial polyposis has a high incidence of adenocarcinomas and some believe that all of these patients will develop a malignancy if they live long enough. Certainly, the common association of polyps with adenocarcinomas of the colon stresses the importance of sigmoidoscopy and barium enema, and early excision to establish beyond doubt the histology.

Curative Surgical Treatment

An acceptable operative procedure for cancer should fulfill the following criteria: 1. Offer a good chance for cure, 2. have a low morbidity and mortality, and 3. maintain as normal a physiologic function as possible.[58]

Use of non-absorbable antibiotics in the intestinal tract preoperatively in conjunction with thorough mechanical cleansing has led to improved healing of the anastomosis, decreased mortality and morbidity and reduced incidence of wound infection.[44] The suggestion that a significant reduction in colonic bacteria might enhance implantation of malignant cells at the suture line is questionable.

Surgical technic has been re-emphasized by Cole.[13,14] Adequate and thorough abdominal exploration determines resectability, metastases, other primary tumors, asociated lesions such as polyps, and other pathology. Minimal manipulation of the tumor, which is covered with a suitable covering such as a rubber sheet, and early ligation of the bowel proximal and distal to the tumor, help to prevent the shedding of tumor cells into the peritoneal cavity and the lumen of the bowel. Early ligation of the lymphatic and vascular trunks at the proximal margins of resection prevents dissemination of dislodged cells into the systemic circulation. All possibilities of contamination of the peritoneal cavity or wound with tumor cells are meticulously avoided. 5-Fluorouracil[49] or half-strength Dakins solution[14] can be injected intraluminally to destroy any tumor cells at the anastomotic line which might be implanted by suture into the bowel wall to serve as a nidus for recurrent tumor. Prior to suturing the anastomosis, all crushed and devitalized bowel is excised to remove an obviously favorable site for recurrence. Cohn[11] uses iodized chromic catgut to prevent seeding of tumor cells in the suture line. He also advocates closed colonic anastomosis and irrigation of the peritoneal cavity with low molecular weight dextran to decrease transplantation of cancer cells in the peritoneal cavity. The high incidence of local recurrence in these lesions and their poor prognosis emphasizes the importance of these preventive measures.[21,28]

The actual extent of the operative procedure should be determined by the lymphatic drainage of the involved area. Since this correlates with arterial supply, the larger the resection, the greater the extent of bowel must be removed because its blood supply is compromised. Tumors of the cecum and right colon are resected with 10 to 20 cm of terminal ileum, the entire ascending colon, and proximal half of the transverse colon in continuity with the omentum, ligating the iliocolic and right colic arteries at their origins. Tumors of the hepatic flexure are resected with this area plus additional transverse colon and the middle colic artery, whereas in tumors of the transverse colon, the entire transverse colon and middle colic artery are resected with omentum. Lesions of the splenic flexure are resected with the distribution of

the middle and left colic arteries along with the omentum. Tumors of the left colon are resected with bowel including omentum from the mid transverse colon to the sigmoid colon at the pelvic floor ligating the inferior mesenteric artery at its origin. Lesions located in the sigmoid colon should have resections which afford at least a 10 cm margin on the tumor distally, taking the inferior mesenteric artery or the superior hemorrhoidal artery at its origin. Any lesion located distally, so that an adequate margin is not obtainable, should have an abdomino-perineal resection.

A procedural problem that often arises in lesions located between 6 cm and 15 cm from the anus is whether a low anterior resection or an abdomino-perineal resection is preferable.[57,58] Certainly, the patient would prefer to have an intact rectum and relatively normal physiologic function. There is, however, the chance of local recurrence at the anastomosis in a tumor which might otherwise be curable. Various groups[13,39,60] have shown the major problem is shedded cancer cells in the bowel lumen which may be implanted at the suture line and the greater the distance from the lesion the fewer cells are present.

It has been documented that distal intramural spread in the bowel wall does not extend beyond 4 cm in curative lesions.[33] Dunphy[18] and McKittrick[60] believe, therefore, that an anterior resection in a potentially curable lesion should be performed only when there is a 10 cm margin distally.

Some physicians report good results with the pull-through procedures, which preserve the anal sphincter.[56] Most surgeons, however, believe that lesions amenable to this procedure could be performed as anterior resections while lower lesions should have an abdomino-perineal resection.

Some surgeons advocate extensions of the previously described operative procedures.[2,3,48] Their purpose is to remove a larger area of lymphatic drainage in hope of improving the overall results. The most popular extensions are ligation of the inferior mesenteric artery at its origin from the aorta, and an aortoiliac node dissection in lesions of the left colon and rectum. Advocates admit to difficulties such as prolonged operative time, more technical problems, and greater likelihood of insecure blood supply to the anastomosis, but state that with proper selection of patients, there are few, if any, significant differences in the complication rates. Bacon[3] believes this more extensive procedure to be contraindicated only in the patient who is a poor risk, markedly obese, or age 65 or older. Results to date are conflicting[2,3,16,34,48] but it may be concluded that in lesions with regional lymph node metastases, these extensions of the usual operative procedures seem beneficial.

Studies of lymphatic drainage of the rectum up to and including the 4-inch level above the anus in females, clearly demonstrate that the posterior vaginal wall, female reproductive organs, and iliac nodes are all involved in the lymphatic drainage of tumors in this area.[5,20] The removal of these structures is an extension of our usual contemporary operative procedure. More recently, certain surgeons have advocated routine bilateral oophorectomy in all women with colorectal malignancies because of the high incidence of recurrence in these organs (10-15%).[4,50]

Wangensteen has employed "second-look" operations in several abdominal malignancies with the most encouraging results in colon carcinomas.[32] At approximately six months after the initial operation, while asymptomatic and without clinical evidence of residual carcinoma, patients with lymph node metastases undergo re-exploratory operations. These are continued until no residual malignancy is found. The advent of a negative exploration results in no further operations. Friese[26] has combined re-exploration with partial hepatectomy in certain cases, with isolated hepatic metastases.

The extended radical procedures pioneered by Brunschwig[6] and Bricker[7] are useful in highly selective cases. Obviously, experience and technical skill are essential; however, the most critical decision involves the judgment of indications for these operations. The large, locally invasive, slowly growing tumor with no evidence of distant metastases is the lesion most amenable to this extended procedure.[9]

The patient with perforative carcinoma of the colon should be treated by resection if feasible.[17] Surprisingly good results have been reported in patients who have undergone definitive surgical resection at initial diagnosis. Overall condition of the patient might well preclude any definitive resection.

Recurrent colorectal carcinoma should be approached aggressively if the lesion is localized. Particularly hopeful is the early anastomotic recurrence found within 18 months of initial surgery on checkup examination of an asymptomatic patient.

Preoperative Irradiation

In the past few years there have been several reports of beneficial effects in the treatment of colorectal carcinomas using preoperative irradiation. At Memorial Center for Cancer and Allied Disease,[45,53] there was no apparent effect on survival rates of patients with carcinoma of the rectum without lymph node metastases; however, patients with lymph node involvement were benefited. The five year survival rate without preoperative irradiation was 23 percent, and with irradition was 37 percent. The ten year survival rates were 10 percent without irradiation and 27 percent with irradiation, even more striking statistics. The average tumor dose was calculated to be not over 2000 roentgens administered with an orthovoltage machine. More recent reports using supervoltage equipment and higher doses continue to be encouraging and certainly merit further evaluation and clinical trials.

As recently outlined, preoperative radiotherapy in cancer is theoretically advantageous because it destroys cancer cells, devitalizes cells which might spread and produce metastases, converts a situation from inoperable to operable, decreases probability of local recurrence and improves tumor immunity.

Disadvantages are: adequate pathology and total extent of tumor may not be known, delay may increase difficulty of surgical procedure and may increase postoperative morbidity and mortality.[41]

Certainly, it is not yet clear which patients should be treated. Current observations[22,23] suggest that supervoltage preoperative irradiation is beneficial in the following situations: a patient with a large localized tumor which is unresectable or unlikely to be cured by resection, a patient whose associated diseases are so severe as to preclude surgery, and a patient with a localized recurrence.

Chemotherapy

The administration of adjuvant chemotherapeutic agents during or immediately following surgery, to destroy circulating tumor cells, is a most attractive theory. Unfortunately, no drug has yet proven effective in altering the survival rates of colorectal adenocarcinomas. Because most chemotherapeutic agents are potent systemic toxins, it is inadvisable to use them as an adjuvant to surgical resection in potentially curable lesions of the colon and rectum until beneficial results are definitely reported.[19,37]

Certainly in the palliative treatment of colorectal malignancies, 5-Fluorouracil has a definite place.[12,38,40] It is well established that with this drug, objective regressions occur in 20 to 25 percent of patients.[46] A closely related analogue, 5-fluoro-2-deoxyuridene (FUDR), is currently being studied with the hope that it might be even more effective. Various methods of administration are suggested. Sullivan and Watkins[54,55] use continuous arterial infusions through hepatic artery catheters for liver metastases

with temporary, but gratifying, results. Rob[47] reported on the intralymphatic injection with radioactive colloidal gold. Rousselot[49] advocates the intraluminal injection of the bowel at the time of surgery.

Administration, however, is usually intravenous. Recent reports[1] have led us to decrease the dosage from 15/mg/kg/day to 12/mg/kg/day intravenously in 5% D/water over several hours for four consecutive days and then decrease the dose further to 6 mg/kg/day administered every other day for an additional seven doses unless toxicity supervenes. Manifestations of toxicity are gastrointestinal symptoms and bone marrow depression. We are impressed with the lack of side effects with this program.

Several reports[46] indicate that 5-FU may increase radiosensitivity. Certainly, combinations of chemotherapy, radiotherapy and surgery seem to be beneficial in treating particular selected cases.

Other drugs such as methotrexate, Thio-TEPA and mithromycin are being used, but 5-FU seems to be the most effective to date in adenocarcinoma of the colon and rectum.

Palliation

Surgery, chemotherapy and x-ray have a definite place in affording relief to the patient with incurable carcinoma of the colon or rectum.[42] Primary resection with anastomosis is the procedure of choice. Colostomy is a miserable procedure to impose on the patient in the terminal stage of widespread malignancy. However, colostomy is definitely indicated in the obstructed patient who is not in the terminal phase of his disease.

Judicious use of irradiation or chemotherapy, principally in the form of systemic 5-Fluorouracil, is extremely helpful. Nerve blocks and certain neurosurgical procedures may be necessary. Of greatest importance is the moral support the physician should give to the patient and his family during this critical period.

Results

The overall 5-year survival rate for patients with adenocarcinoma of the colon and rectum is 30-40 percent. This remains a depressingly static figure. In some series there has been definite improvement in results, but in many a plateau has been attained and results remain unchanged.[6,24,25,27,29,51,59]

Summary

Colorectal carcinoma is second only to skin cancer in prevalence of malignancy. To improve results of existing treatment, earlier diagnosis and therapy is mandatory. Each case must be carefully evaluated on its own merits before therapy is initiated. Whether to extend the operation or utilize preoperative irradiation and adjuvant chemotherapy are all considerations. Certainly, there is no place for a rigid approach, but rather, "fit the treatment to the patient, not the patient to the treatment."

Editor's Note: The bibliography may be obtained from the author.

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Goodpasture's Syndrome

A Cause of Fatal Pulmonary Hemorrhage in a Nineteen Year Old Male

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Goodpasture's Syndrome should be considered in all cases of hemoptysis in younger persons, especially if urinary evidence of glomerulitis is present.

GOODPASTURE'S SYNDROME is a disease of unknown etiology characterized by diffuse pulmonary hemorrhage and glomerulitis.[1-19] Many patients exhibit diffuse vasculitis and die of progressive renal insufficiency.[20] An immune mechanism has been postulated but immunosuppressive therapy has not been helpful.[21-22]

Case Report

A nineteen year old white male was admitted to the Bedford County Memorial Hospital on December 21, 1965, because of massive hemoptysis of several hours duration.

Past history: He had previously been well except for a "slight cold" for two to three weeks. He had taken symptomatic medicines for this without much relief. The past several days he began coughing frequently and the day prior to admission began to expectorate mucoid-bloody material. This increased in severity and became more bloody. He denied chest pain prior to the onset of coughing, sweats, chills, fever, occupational exposure to toxic chemicals, weight loss or wheeze.

Systems review, family history and social history were non-contributory.

Physical examination: T 103, P 100, R 24, BP 116/60. He was a well developed, well nourished nineteen year old muscular white male in acute distress with paroxysms of coughing. Skin: hot and dry. No rash. HEENT: eyes normal. Posterior pharynx reddened with bloody mucus. Neck: supple. Thyroid normal. CVS: R and R normal. No murmurs. No cardiac enlargement. Lungs: equal expansion with slight dullness to percussion in both bases posteriorly. Both lung fields filled with medium moist rales. Abdomen: LKS not felt. No masses or tenderness. Genitalia: normal male. Extremities: normal. Neurological: DTR's equal and active. No abnormal reflexes. Sensorium clear.

Laboratory studies: Hgb 9.5 grams. HCT 34, WBC 12,250 with 77 polys, 13 bands, 1 Eos, 6 lymphs and 3 monos. Peripheral smear revealed the red blood cells to be hypochromic, platetlets increased (327,000) and the white cells to be normal. Urine yellow, hazy, acid, S.G. 1020, 3+ albumin, negative sugar and acetone. Micro—many red blood cells, 7-8 WBC and 1-2 epithelial cells per h.p.f. Urine positive for blood. BUN 26.8 mgm. % Coombs test negative. Prothrombin time 14 seconds (C=13 sec.). Blood type AB+. A sputum culture later grew out normal flora.

X-ray: P-A and lateral chest films revealed ill-defined bilateral pulmonary infil-

tration compatible with bronchopneumonia and/or aspirated blood (Fig. 1).



Fig. 1. Chest x-ray.

The patient was placed at bed rest, started on symptomatic treatment and Keflin. He began to feel somewhat better but still continued to cough up large amounts of blood. His ventilation was adequate, however, and no cyanosis was observed. Twenty-four



Fig. 2. Lung.

hours after admission his hemoptysis increased and his hemoglobin was noted to be 8.0 grams. He was started on oxygen and transfused with one unit of blood. Over the next few hours he became steadily worse with increasing shock, respiratory difficulty and deep cyanosis. A tracheotomy was performed and suction carried out with some results but the downhill course continued and the patient expired approximately thirty-six hours after admission.

Autopsy Examination

The body was that of a well developed, well nourished muscular nineteen year old white male. The head, eyes, ears, nose and throat show no abnormalities other than a considerable quantity of blood oozing from the nose and mouth. The upper extremities were normal. There was a recently placed tracheostomy incision in the midline

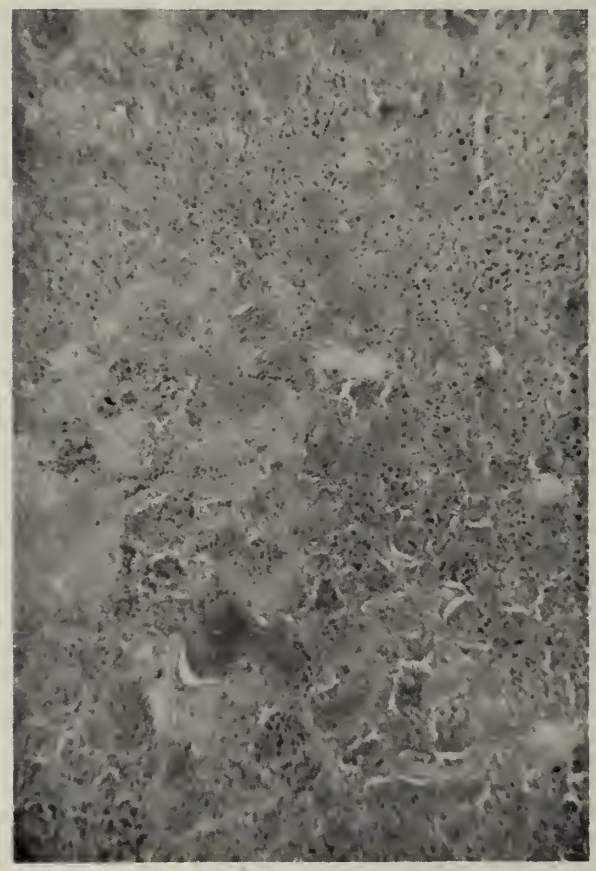


Fig. 3. Lung H & E stain x 40.

of the lower part of the neck. The chest, abdomen and external genitalia were unremarkable. The lower extremities showed no lesions. There were no areas of petechiae or ecchymoses noted in the skin.

On reflecting the sternum the lungs were noted to be diffusely firm and red in color. No hemorrhages noted about the pericardium. There was no fluid in either pleural space. The abdominal organs appeared grossly unremarkable.

The heart was normal with no congenital abnormalities noted. The coronary arteries were normal. There was slight dilatation of the cavity of the left ventricle.

A tracheostomy incision was noted in the anterior portion of the trachea 4 cm. below the thyroid cartilage. The trachea and larger bronchi were completely filled with bloody froth.

The lungs weighed in excess of 2500 grams. The parenchyma showed diffuse hemorrhage with widespread consolidation throughout. The parenchyma was rubbery



Fig. 4. Lung H & E stain x 100.

and had the appearance of advanced red hepatization. The only area that was not affected in this manner was the right middle lobe. The major bronchi were traced out with no obvious bleeding points seen. There were some enlarged lymph nodes noted at the hilus (Fig. 2).

The gastrointestinal tract was normal.

The liver weighed 1600 grams and showed moderate congestion.

The adrenals showed marked lipid depletion.

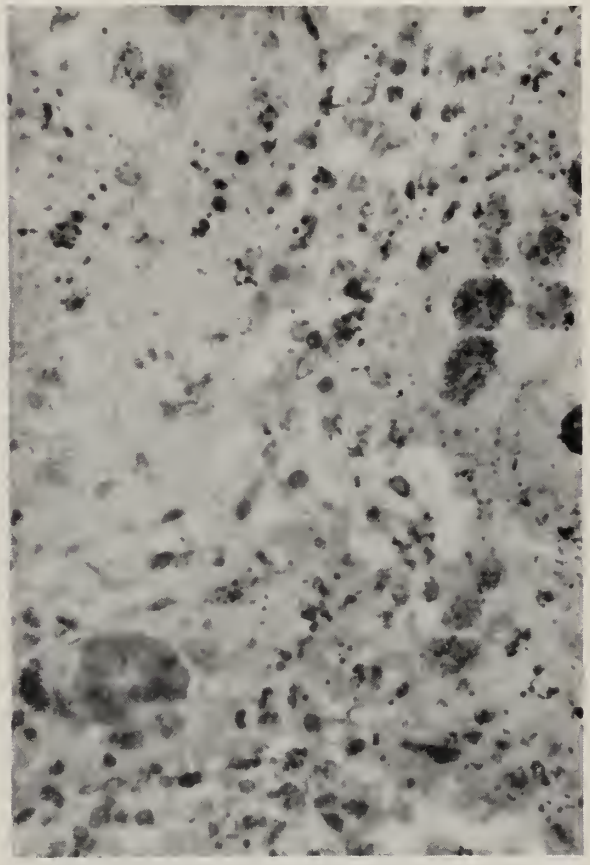


Fig. 5. Lung H & E stain x 450.

The kidneys weighed 165 grams each. The parenchyma showed scattered petechiae and the capsules stripped with ease. Ureters and bladder were normal as was the remainder of the gross examination.

Microscopic Description

The heart was found to be normal. There was diffuse widespread hemorrhage, old and recent, in the lungs. There were many intact red cells with hemosiderin-laden macrophages and extensive pulmonary edema with many foci of inflammation with alve-

olar necrosis. There was no evidence of vasculitis or any other pre-existing process (Figs. 3, 4 and 5).

A bronchial lymph node showed marked chronic inflammation. The trachea, spleen and adrenals were all normal. In the kidney there was moderate swelling of glomerular tufts with focal basement membrane thickening. Scattered glomeruli showed fibrinoid necrosis and there were many red cell casts in tubules with proteinaceous casts in other areas (Figs. 6, 7 and 8).

The liver, pancreas and bone marrow studies were normal.

Final Anatomic Diagnosis

1. Intra-Pulmonary Hemorrhage, Acute, Severe (Goodpasture's Syndrome);

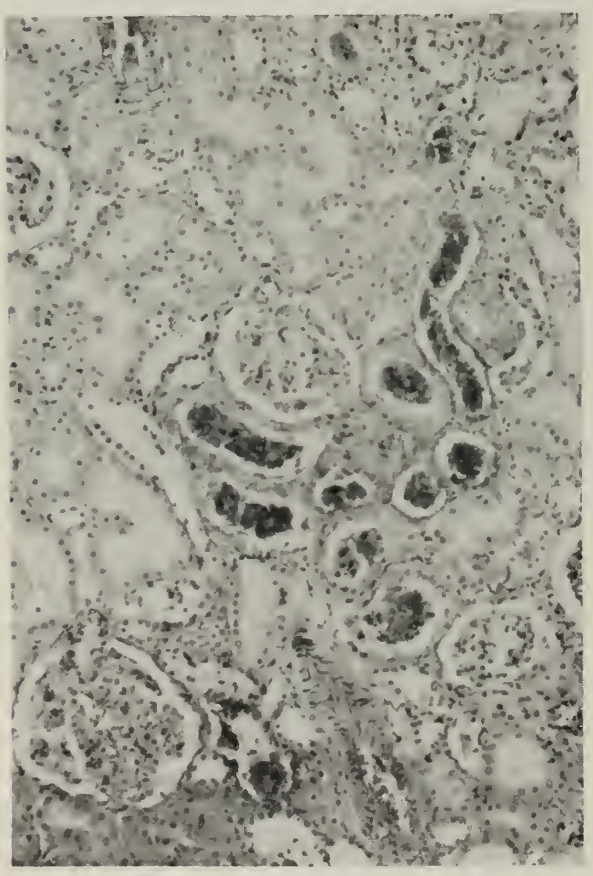


Fig. 6. Kidney H & E stain x 100.

2. Acute Glomerulonephritis;
3. Post Operative State, Recent Tracheostomy.

Discussion

The course of disease in this young male was not over three weeks and he died principally of suffocation due to the overwhelming pulmonary involvement. This fulminant

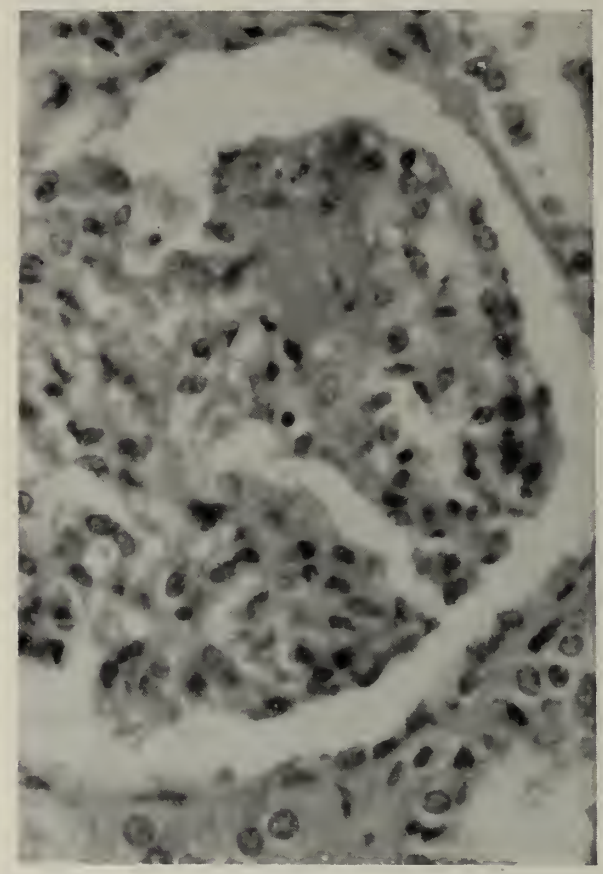


Fig. 7. Kidney H & E stain x 450.

course is somewhat unusual and raises the usual questions of etiology and treatment. This disease should be considered in all cases of hemoptysis in young persons, especially if urinary evidence of glomerulitis is present. The rapid onset and course make the tissue changes less striking than some previously reported cases but still the lung findings are impressive.

Treatment has been generally ineffective in this disease and has consisted mainly of steroids, azathoprine, blood replacement and general supportive measures.

Summary

A young man developed overwhelming pulmonary hemorrhage with glomerulitis

and expired of suffocation. Goodpasture's Syndrome should be considered in all cases of hemoptysis in younger persons, especially if urinary evidence of glomerulitis is present.

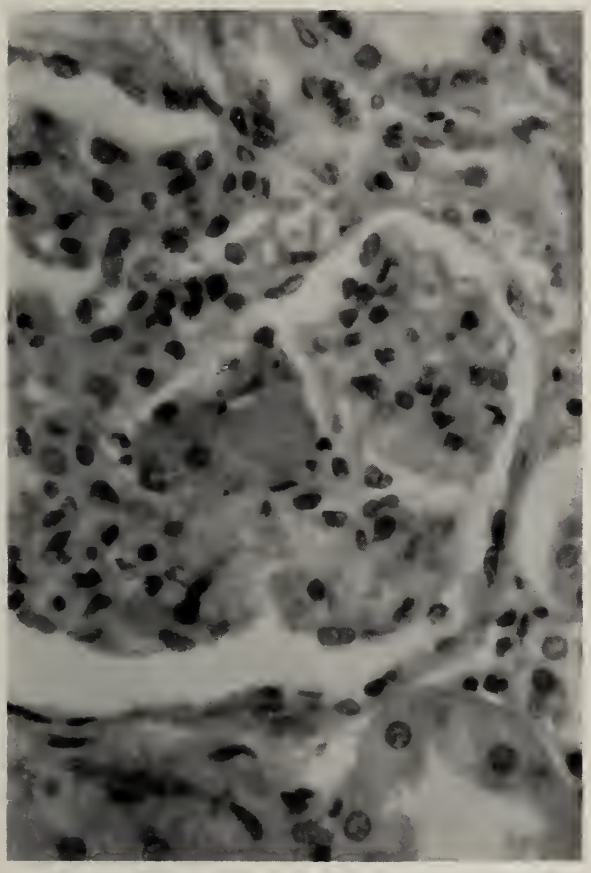


Fig. 8. Kidney H & E stain x 450.

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Jefferson's School of Medicine at The College of William and Mary in Virginia

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In 1779 and twice since, attempts were made to establish a school of medicine at the College of William and Mary. All three attempts were interrupted by historical circumstances, some of which were political.

AT THE TIME OF THE REVOLUTION, William and Mary was the wealthiest and most influential College in the Colonies. It was the major intellectual resource of Virginia. Although wealthy and under royal patronage, the College chose to risk its material substance in support of the Revolution and to oppose the policies of the British Crown. The College subsequently took an active part in the events which accompanied the Revolution and the founding of the Republic.

The roll of fame of William and Mary includes Alumni who probably exerted greater influence in forming the nation than the alumni of any other institution. Peyton Randolph was the first president of the Continental Congress in 1774. Thomas Jefferson authored the Declaration of Independence. John Tyler, Jr., carried through the Virginia legislature the proposition for the Annapolis Convention of 1787. George Washington consolidated the administration of the new Constitution and assured its workability. John Marshall settled the construction of that Constitution. The contribution of these and many other alumni of that day has occasioned the College to be referred to as the Alma Mater of a Nation.

William and Mary became the nation's first full-fledged University—a fact achieved largely by a major curricular reorganization undertaken in 1779 by Thomas Jefferson. This University status was attributed, in part, to the curriculum in medicine.

It is clear from his writings that Jefferson recognized Medical Education as a part of University Education. He said: ". . . you may promise yourself everything—but health, without which there is no happiness. An attention to health, then, should take place of every other object." In keeping with William and Mary's high traditions of academic innovation and excellence, Jefferson established a School of Medicine in his reorganization of the College in 1779.

The 1779 reorganization of the College of William and Mary, which inaugurated the University status, not only established a School of Medicine, but also a Law School (now Marshall-Wythe School of Law), and a School of Modern Languages. The School of Medicine was the third to be established in the Colonies, and out-dated Harvard Medical School by three years. The first Medical School founded in the Colonies was established in 1765 at the College of Philadelphia, now the University of Pennsylvania. The second was organized in 1767 at King's College, now Columbia University, but teaching was suspended in 1776.

Prior to the 1779 reorganization and the establishment of a School of Medicine, which included both Anatomy and Chemis-

try studies, the College served other important functions. As an establishment of the Anglican Church, one of its functions was the preparation of ministers for Virginia Churches and missionaries for the training of American Indians. It also sought to prepare men for other learned tasks of life, and particularly public men who were adapted to the arts of statecraft. By the time of the Reorganization the College had possessed a full faculty of regular professors for some years, and it is clear that one of the great dreams of Jefferson's early years, therefore, was the transformation of an already stable William and Mary into a great national University. This, it may be said, was the larger purpose in 1779.

It may be recalled that in October, 1776, Jefferson surrendered his seat in the Continental Congress, and re-entered the Virginia Legislature to devote his time to the revision of Virginia laws and to codification of the great principles of political freedom for which the Revolution was fought.

Jefferson's Plan, particularly with its emphasis on medicine, was a definite attempt at secularization of the William and Mary curriculum. It met strong and heady opposition. In his *Autobiography*, Jefferson states that he drew his detailed plan (Bill no. 80) in order "to amend the constitution of William and Mary, to enlarge its sphere of science, and to make it in fact a University." He said:

> ". . . its (William and Mary's) constitution, organization and scope of science were derived from its Charter. We thought, that in this subject a systematical plan of general education should be proposed, and I was requested to undertake it. I accordingly prepared three bills for the Revisal. . ."

Bill no. 80 was one of the three bills.

The religious aspects of the bill, however, and the jealousies against supremacy of the Anglican Church in the preparation of ministers, delayed results. Jefferson took direct action on becoming Governor in 1779:

> "On the 1st of June, I was elected Governor of the Commonwealth and retired from the Legislature. Being elected also one of the Visitors of William and Mary College, a self-electing body, I effected during my residence in Williamsburg that year, a change in the organization of that institution of abolishing the Grammar School, and the two professorships of Law and Police, one of Anatomy, Medicine and Chemistry, and one of Modern Languages."

It must be noted that Jefferson was strongly influenced at this time by the ideas of a Frenchman, Quesnay de Beaurepaire, who had proposed the establishment of an Academy of Sciences and Fine Arts with French professors, masters and artists at Richmond, Virginia. The concept of the Academy was that of a graduate school designed to teach and provide research in various fields, including anatomy, chemistry, and natural history. Quesnay was encouraged in 1778 by the lieutenant governor of Virginia, John Page, and Jefferson supported him. A building was erected but the project collapsed.

Although the scheme failed, it was not without fruit. When Jefferson was developing his own ideas regarding the future of William and Mary, particularly with regard to Science, it is evident that he had Quesnay's concepts in mind.

The story of the School of Medicine established in 1779 would lack its central focus if mention of James McClurg, the College's first teacher of Medicine, were overlooked. It was the reorganization, in fact, which brought McClurg to the newly created Chair of Anatomy and Medicine. For three years McClurg held this position and then moved to Richmond. Since the records of the College have been lost for this period, it is impossible to learn how much teaching

was done by McClurg or why the chair was discontinued when he left.

McClurg was a man of wealth and prominence and his presence on the William and Mary Faculty was, indeed, significant. As a boy he studied at William and Mary with distinction. He was particularly distinguished for his high scholarly attainments in classical learning. The fact that he was graduated in 1762 and studied simultaneously with Jefferson, who also finished his work at the College at that time, should not be overlooked.

Shortly after graduation, McClurg decided upon a career in the medical profession and took up residence at the University of Edinburgh, where he won the affection of such esteemed men in the profession as Cullen and Black. McClurg greatly justified these friendships by the production of a thesis, *DeCalore*, that is considered both profound and original. It earned for him an admirable reputation among scientific friends.

McClurg subsequently devoted several years to post-graduate medical studies in London and Paris. It was in London that he published his celebrated essay, *Experiments Upon the Human Bile and Reflection on the Biliary Secretion*. This work, published when McClurg was only twenty-six years old, aroused considerable notice, was reckoned as a valuable contribution of the science of medicine, and was translated into a number of foreign languages.

The work was introduced by an essay on medical reasoning in which he pointed out, briefly, that medical science had been subject to change from the earliest times—fortunately progressive—and that the quarrel between the dogmatists and empiricists was an ancient one. He then attempted to show that the quarrel has been salutary to progress, and that there was something of truth on both sides—a belief which convinced him of the general usefulness of new theories. Having established this thesis, McClurg introduced his own theory.

Nothing was more calculated to give him prestige in his profession in the Colony of Virginia than to have been the author of a theory in an age peculiarly deferential to this type of intellectual exercise. Couched in fine English, ornamented by frequent classical quotations, and conveying all the persuasion of a syllogistic argument, it was irresistible and was destined to propel him toward an institution such as William and Mary, where learning, combined with intellectual innovation, was the proved essence of curriculum.

Heralded by his theory, McClurg returned to Virginia amidst contemporaries who included in their number Dr. James Munsion Galt, Drs. James and William Carter, Dr. William Pasteur, and others. For ten years McClurg made his residence in Williamsburg and during this time acquired considerable reputation. During this period —1779—McClurg was appointed Professor of Anatomy and Medicine at William and Mary.

Concerning this appointment and its significance, which was effected by Mr. Jefferson's reorganization and election to the Board of Visitors, John Brown, a student at the time of the appointment, wrote. "William and Mary has undergone a very considerable Revolution; the Visitors met on the 4th Instant and form'd it into a University. . . " The chair of medicine, which helped to achieve such University status, was discontinued in 1783. As stated, little is known about what transpired during its brief years of existence.

We may best glean its value and contribution from a retrospective comment made by President James Madison shortly after the Chair's discontinuance. Madison, as College President, expressed his lament at the small attention—even neglect—paid to science since Dr. McClurg's resignation from the College Faculty in 1783. He also commented that:

> The Professorship of Chemistry . . . has not been actually abolished; but after Dr.

McClurg left us, two professorships of Humanity were instituted in its stead.

Although we may not summarize what transpired during McClurg's years as a Faculty member at the College, we may deduce some of his teaching practices from the more general beliefs we held. It must be remembered, first, that McClurg was an exception to the general run of doctors of his time, in that he did not possess or employ surgical instruments and did not own an apothecary shop. This early resolution to woo only one branch of medical science was adhered to throughout his life.

Yet in the training of the physician, McClurg thought there should be no separation of the sciences. He considered every branch of science as kindred and capable of mutual illustration. "The sciences," said McClurg

> "like the graces, march hand in hand, and nothing would be more vain than an attempt to pursue any one of them separately."

McClurg also opposed the separation of physic and surgery as unnatural, and insisted that it would be for the advantage of both to unite them. Yet he admitted, as far as simple dexterity is concerned, that the chirurgical art has been improved by the separation. Though McClurg probably did some teaching at William and Mary, it must be kept in mind that his broader interests were evidenced to be more academic than professionally practicing ones. His concern, noted in his epitaph, remained "the cultivation and diffusion of medical literature in the United States."

The discontinuance of McClurg's Chair of Anatomy and Medicine brought an end to Jefferson's School of Medicine at the College of William and Mary—at least as he himself had created it. His concepts endured, however, and that which he had envisioned was to be revived on two successive attempts. One revival occurred during Jefferson's very last years but it was without Jefferson's support, for larger political reasons. It nonetheless was an outgrowth of his own ideas on medical teaching.

This attempt to re-introduce medicine at William and Mary occurred in 1824. The fortunes at the College were at low ebb. The patronage of the school had seriously fallen off, and only radical measures promised to restore lost prestige. Dr. John Augustine Smith, President of the College, proposed the bold plan of moving the institution to the state capital in Richmond. Smith conceived a great University with departments of theology and medicine, and he went before the legislature with his plans. Part of his testimony reads:

> A medical school would be organized and attached to the College, and the theological school might be reunited to us. . . (This) would give utility, dignity, and importance to the institution . . . and by increasing the reputation of the College, thus augment indirectly our fortunes.

The scheme was well thought out and would probably have meant much for medical education in Virginia. It was endorsed widely by physicians throughout the State. The plan, however, ran athwart the maturing plans of Jefferson for another university at Charlottesville, and thus incurred his opposition and failed.

Joseph C. Cabell, long-time correspondent with Jefferson, wrote the aging statesman (now 81):

> A scheme is now in agitation at the place, the subject of which is to remove the College of William and Mary to the city of Richmond. . . . The clergy, the Federal party, the metropolis, and probably the faculty of medicine throughout the State, will advocate the removal. . . . The hostile party in Richmond and the College aim decidedly at a great institution connected with a medical school.

Jefferson recognized the full significance of the situations. He knew well that it

meant the ultimate defeat of his own cherished project of a state university. He replied to Cabell, on December 22, 1825: "The proposition to remove William and Mary College to Richmond, with its present finds, and to add to it a medical school, is nothing more nor less than to remove the university also to that place." Such opposition was sufficient to kill the project, and the proposals of Dr. Smith were defeated.

Seventeen years after Dr. Smith's efforts, the teaching of medicine was again attempted at William and Mary. Jefferson was no longer living to exert his personal influence, although the influence of his ideas remained to underscore the third and final effort to introduce medicine at the College.

Dr. John Millington, engineer, scientific writer, and teacher, was professor at the College and the central figure in William and Mary's last attempt to teach medicine. Although Dr. Millington acquired his M.D., he never practiced medicine but devoted himself to engineering and teaching. His teaching background included extensive lecturing on natural philosophy, mathematics, and astronomy. In 1835, when he accepted the Chair of Chemistry, Natural Philosophy, and Engineering at the College, he was well acclimated to the curriculum needs in medicine at that time. The College catalogue in 1841-42 bears out this realization:

> As nearly all the Medical Colleges of the union require that a student shall have studied medicine with some practitioners for two years before he offers himself for their instruction and for graduation, Professor Millington undertakes a class in the department of science.

Referring to this innovation in Virginia, Robley Dunglison wrote in the medical journal of which he then was editor: "We find by the Annual Catalogue of this old and respectable institution (William and Mary), that Professor Millington undertakes a class of medical instruction, for which he possesses ample means of illustrations. The subjects taught in the first session are anatomy, philosophy, materia medica and pharmacy continued and concluded. The textbooks are the same as those used in the principal medical colleges." Dunglison's final statement, particularly, offers proof of medical teaching at William and Mary.

Like the first two attempts, the third dissipated quickly. A faculty quarrel precipitated undue animosity. All but one faculty member subsequently resigned from the College in 1848 and Dr. Millington, among them, terminated his connections with William and Mary at that time. The College closed for the session 1848-49.

We may conclude that the establishment of medical schools in early America would not be complete without an historical realization of medical programs at the College of William and Mary; that this realization is relatively unrecognized, but not of recent origin; that Thomas Jefferson was especially instrumental in inaugurating a School of Medicine at the College of William and Mary; that this School of Medicine was vital in assisting William and Mary in its conversion to larger university status; that Jefferson, in his effort to reorganize the College with a School of Medicine, believed medical education to be an integral part of University Education; that William and Mary was particularly adaptable to medical education by its academic excellence and traditions of curricular innovation; that curricular instruction was most probably directed along lines of research and graduate techniques, rather than practical physics and surgery; that three attempts to introduce medical teaching at the College were undertaken, in total, during its long history; that the latter two attempts were intended most likely to follow the curricular organization of other already existent Medical Colleges; that all three attempts were interrupted by historical circumstances, some of which were political; and that for the period of their existence, all attempts reflected Mr. Jeffer-

son's original concepts for a School of Medicine at the College of William and Mary. Their failure to succeed, therefore, constitutes, in part, the unfulfilled vision of Jefferson in the initial establishment of the School of Medicine in 1779.

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Let's Reminisce!

MEDICAL SOCIETY OF VIRGINIA—We do not know that the Society ever had a more harmonious and profitable session than that recently adjourned, and which convened at the Fauquier White Sulphur Springs, September 13th. The attendance was good—about fifty—although the time lost, and the inconvenience in getting to the Springs by reason of want of connection of trains, and the frequent change of cars, was very great. For instance, passengers from this city, going by the most direct route—via Gordonsville—have to remain at that depot five hours. Then, again, at Orange Courthouse, change cars; at Warrenton Junction, change cars again for Warrenton; and at this latter place, change to stages for the Springs, some ten miles off—all of this to travel a distance from Richmond of about 130 to 140 miles. But the attention given by the proprietor of the Springs and his clerks to the entertainment of his guests, more than compensated for every inconvenience. (Virginia Medical Monthly, October, 1882)

The Great Idealist

The Life and Times of Dr. Ennion G. Williams

FREDERICK J. SPENCER, M.D.
Richmond, Virginia

A great humanitarian, Dr. Ennion G. Williams, as first Commissioner of Health, directed the State Health Department for 23 years, from its beginning until it was an efficient, well organized department.

THE YEAR 1874 was conspicuous for its uneventfulness. In England, Benjamin Disraeli became prime minister for the second time while in Switzerland an International Postal Congress debated mail services which were inaugurated next year. The most interesting event in the United States was that William Marcy "Boss" Tweed, having defrauded New York City of some $30,000,000 with his Tammany colleagues, was sentenced to 12 years imprisonment by the action of irate citizens. In Virginia, however, two events of importance in public health occurred. First, a 23-year-old doctor from Gloucester County entered the Medical Corps of the United States Army after graduating from the University of Virginia at the age of 17. His name was Walter Reed and his work in yellow fever was to culminate in its application by General William Crawford Gorgas in the building of the Panama Canal. The other event occurred in Richmond on 31 January 1874 when a son, christened Ennion Gifford Williams, was born to Mr. and Mrs. John Langbourne Williams. This baby was destined to become the first Commissioner of Health for the State of Virginia.

Ennion G. Williams' father was a prominent Richmond business man descended from Irish ancestors who migrated to America in the 1820's. His mother was descended from the Dandriges and Randolphs, both of whom were closely associated with George Washington. After a preliminary education at McGuire's University School in Richmond, Ennion G. Williams entered the University of Virginia and graduated with the degree of Doctor of Medicine in 1897. He interned at Willard Parker Hospital, New York, which was then one of the foremost infectious disease hospitals in the world. Moving to Philadelphia, he worked in the Polyclinic Hospital whence, after a short visit to Munich, Germany, he returned to Richmond. In his native city, Dr. Williams was a pioneer of radiology, both diagnostic and therapeutic, becoming vice president of the American Roentgenological Society in 1904, nine years after Wilhelm Konrad Roentgen had demonstrated the first practical use of x-rays. Dr. Williams' interest in infectious diseases was maintained by his appointment to the Chair of Pathology, Bacteriology and Histology in the Medical College of Virginia in 1901. His association with the Medical College of Virginia continued as Professor of Preventive Medicine from 1916 until 1923.

Dr. Williams married Anna Heath Lassiter, daughter of Dr. Lassiter of Petersburg, in 1902. His courtship required arduous journeys by streetcar between Richmond and Petersburg and, on one occasion,

The Walter Reed Lecture, 1966, presented before Section on History of Medicine, Richmond Academy of Medicine.

after missing the last streetcar he was forced to walk home, arriving in time for breakfast. Dr. Lassiter, who was in ill health at the time, was impressed with his future son-in-law and did everything he could to hasten the marriage. It is said that Dr. Williams appeared at his sister's house one day and asked her to go and buy him a collar and prepare him for marriage. This act was the foundation of a family joke that his sister was the "best man" at his marriage.

During the early part of his career in Richmond, Dr. Williams was active in civic and medical affairs, becoming the treasurer of the Richmond Academy of Medicine in 1903, and its president in 1907. He was elected to the Richmond City Council in 1905. His intent to reform the health services of the city was soon evident, and he was directly responsible for obtaining funds for the appointment of the first city bacteriologist, Dr. E. C. Levy, who had also served at Willard Parker Hospital. Dr. Williams campaigned strongly for the passage of the "Baker Bill", a measure introduced in the General Assembly by Captain William W. Baker of Chesterfield County for the reorganization of the Virginia State Board of Health. This was passed by the State Legislature in 1908 and Dr. Williams was asked to be the first Commissioner of Health at a salary of $3600 a year, possibly 1/3 of his income from private practice. He accepted the position and directed the Health Department for 23 years. During this time he was president of both The Medical Society of Virginia and of the Conference of State and Provincial Boards of Health in North America, the forerunner of the present State and Territorial Health Officers Association. He was reappointed by the next six governors, the last being Governor Byrd in 1928. Although suffering from ill health, he was still active in 1931 as shown by an editorial in a local paper on 26 February which described him investigating sanitary conditions in the oyster industry on the Eastern Shore. Dr. Williams, however, was unable to be at the Board of Health meeting on 16 March 1931 and suffered a stroke during his sleep on 19 May, dying at the age of 57 on 6 June 1931.

Ennion G. Williams *was* public health in Virginia for 22 years and developed a tradition which began at Jamestown in 1610. In this year, a law was passed dealing with sanitation stating in part "nor shall anyone aforesaid, within less than a quarter of one mile from the Pallizadoes, dare to doe the necessities of nature." Local boards of health undoubtedly were formed and disbanded to deal with epidemics during the eighteenth century and it is possible that Petersburg may have had the first permanent city board of health in America, although the records substantiating this were destroyed in the Civil War. In 1871, James Lawrence Cabell, Professor of Surgery, University of Virginia, promoted a petition to the General Assembly at the second annual meeting of The Medical Society of Virginia asking for the formation of a board of health in the State of Virginia. The legislature passed a bill to this effect in 1872 but eliminated the $4000 for the secretary's salary and the expenses of the board which had been suggested by Dr. Cabell. Dr. Cabell was also president of the first National Board of Health which was extant between the years of 1879 and 1883.

The Virginia State Board of Health met for some years at the personal expense of its members and then disbanded. In 1893, being alarmed at reports of cholera, Governor McKinney revived the Board of Health at the request of the Richmond Chamber of Commerce. The Chamber of Commerce then contributed $300 towards the publication of a cholera pamphlet, the first constructive move by the Board of Health. This action unwittingly set a precedent for one of Dr. Williams' most cherished projects, the Virginia State Health Department's *Health Bulletin*. In 1896, the General Assembly appropriated $2000 for the Board of Health and increased this to $5000 in

1899 when smallpox threatened to invade the State.

Thus, at the turn of the century, the State Board of Health was established as an active government agency. At this time, of course, the population was more than half rural and the lack of sanitation was reflected in the devastating epidemics of infectious diseases, particularly typhoid fever, which ravaged the State. Tuberculosis and malaria were also endemic and epidemic. It was the attack on the formidable mortality occasioned by these communicable diseases that was led by Dr. Williams in his initial work in the Health Department.

The aims of the new health commissioner were set out in volume 1, number 1 of the *Health Bulletin* in July 1908. His intention, from the beginning, is exemplified in his statement that he wanted to educate the public on the "nature, cause, and means of prevention of such diseases as tuberculosis, malaria, typhoid and to give general instructions in matters pertaining to public health."

Dr. Williams had an interest in tuberculosis which remained with him throughout his career and which may have attracted him to the "Baker Bill", the main element of which was the erection of a tuberculosis sanatorium in the State of Virginia. This was realized in 1909 when Catawba Sanatorium was opened at Roanoke Red Sulphur Springs. Dr. Williams' interest in tuberculosis was certainly maintained by his misfortune in having a son with tuberculosis who recovered after treatment in a State Sanatorium. Dr. Williams also established the category of health personnel known as "Certified Tuberculosis Nurses"—women who had recovered from tuberculosis and, after receiving two years training at Blue Ridge Sanatorium and one year at the Medical College of Virginia or University of Virginia, became eligible for the State Board of Nursing examination. Dr. Williams went to Blue Ridge Sanatorium every year for the graduation of the "Certified Tuberculosis Nurses," continuing this custom until the year of his death when he watched the exercises from his car.

Dr. Williams worked with two pioneers of public health nursing in the State in the persons of Miss Agnes Randolph and Miss Nannie Minor. These individuals were totally different in temperament and it says much for Dr. Williams' tact that he was able to work equally well with both persons. Miss Randolph, who became executive secretary of the Virginia Tuberculosis Association and also director of the Tuberculosis Out-Patient Service in the State Health Department, was an aggressive champion of health services and constantly pursued Dr. Williams, calling him at home on many occasions to promote better health services. Miss Minor, a founder and director of the Richmond Instructive Visiting Nurse Association and later director of the Bureau of Public Health Nursing in the State Health Department, was exactly the opposite to Miss Randolph, being a persuasive and quiet person who achieved her goals by a knowledgeable deference to Dr. Williams and her colleagues. These pioneers of public health nursing, both educated at the Medical College of Virginia, are remembered in the building now known as Randolph-Minor Hall at the College.

Dr. Williams travelled throughout the State in the promotion of health services, often denying himself a vacation to continue his work. He rented a cottage near Catawba Sanatorium for two months during the summer, on one occasion, in the hope that his wife and family would see more of him than if they were to stay in Richmond. Unfortunately he developed mumps and spent a good deal of his time in Richmond while his family was at the cottage.

Dr. Williams was particularly fortunate in having two distinguished members of the Freeman family as colleagues in his early days with the Health Department. Dr. Allen W. Freeman acted as an Assistant Commissioner before becoming an epidemiologist

with the United States Public Health Service. Afterwards he was Commissioner of Health for the State of Ohio and ultimately became Dean of the Johns Hopkins School of Hygiene and Public Health in Baltimore. In 1910, he initiated the hookworm investigation in Virginia, supported by the Rockefeller Foundation, which was to become nationally famous. Dr. Williams also obtained the services of the noted historian and future Pulitzer Prize winning editor of the *Richmond News Leader,* Douglas Southall Freeman, as the first editor of the *Health Bulletin* in the State Health Department. The bulletin was supplemented by the *Virginia Health Almanac* in 1911, the first of many such publications.

In 1912, the General Assembly appropriated funds for the position of a state registrar as a result of a crusade by Dr. Williams for better statistical reporting and in 1913 the first comprehensive vital statistics were made available. The main disease in rural Virginia at that time was typhoid fever and on one occasion it is said that Dr. Williams told the General Assembly that if the money he had asked for were appropriated he would wipe out typhoid fever in the State. In addition to this, he is reported as saying that he would donate one year's salary towards the General Assembly's funds for the typhoid fever campaign. He certainly said that if any part of the budget were not approved it should be his salary rather than the funds for typhoid eradication. His family apparently remained in trepidation for some time, anticipating that they would be sent to the poor house because of his statement. Dr. Williams was a pioneer in improving sanitation in the State, promoting the use of the sanitary pit privy, then the best single method of controlling typhoid fever. Despite a coronary attack in the early 1920's, Dr. Williams continued to travel throughout the State, on many occasions being driven by one of his daughters or another member of the State Health Department. His family was active in public health, two of his sons conducting summer campaigns for communicable disease control, one of his daughters directing the State Health Department's Bureau of Biologics and another daughter acting as an advisory nurse in the same department.

Ennion G. Williams was more than anything else a humanitarian. It is recorded that Frances Richardson, born in slavery and cook in his father's house who was known to Dr. Williams as "Aunt Frances", sought his professional care in her old age. She died in 1902 and as a measure of her appreciation left her life insurance of $468 to Dr. Williams with which he purchased an x-ray machine and a "morsel of radium". As a scientist, he is credited with the x-ray law that the intensity of radiation varies with the square of the distance but, despite these abilities, his main interest was always in public health. He was a man of high integrity and would not use a State car for anything but State business. He and his family would walk one and a half miles to church across the fields in muddy weather rather than use the car which almost certainly would have been accepted as within the prerogatives of the State Health Commissioner. He was a staunch member of the Episcopal church, conducting prayers night and morning and ending them one day with a modified biblical quotation that there was "a time for great things, a time for small things, and time for the streetcar" on hearing the bell outside the house. Although against alcoholism as a public health menace, he was not a teetotaler and always had wine in the house. His father would order a tun (252 gallons) of Madeira wine which had to be transported around Cape Horn to San Francisco and then driven overland to the Williams' home in Richmond where it was distributed in gallon jugs among the various members of the family. He was a traditionalist without being fettered by it and broke with custom by writing the legislation for the appointment of a dentist to the State Board of Health.

Virginia was the first State in the United States to accept dentistry in this manner. He was a politician without any of the ambiguity sometime seen in politics and although a loyal Democrat, declined to campaign for state governor when he was requested to do so on one occasion. Above all he was a tireless worker for the health of the Commonwealth and indulged in few outside interests.

One of Dr. Williams' prized possessions must have been a letter written by the doyen of public health workers in the United States, Dr. Charles-Edward Amory Winslow, Professor of Public Health at Yale University who wrote—

THE BIOLOGICAL LABORATORIES
MASSACHUSETTS INSTITUTE OF TECHNOLOGY

BOSTON, MASS. April 11, 1910

Dear Dr. Williams:

I have just been reading your Health Bulletin for January-February, containing the annual report of your department, and I cannot refrain from writing you a line of congratulation thereon. It is wonderful work you are doing in building up all at once a complete modern department of health. Already the educational work of the Virginia Board is I think the best in the country. Indiana is the only State that ranks with it. You have a wonderful opportunity in the study of rural typhoid, hookworm, and other particularly southern maladies, and it is a great thing for the whole country that you are rising to it so successfully.

Very truly yours,
C.-E. A. Winslow

On his death, a resolution passed by the Richmond Academy of Medicine described Dr. Williams as having "the gallantry of Sir Philip Sydney, the sacrificial modesty of Walter Reed, the determination to conquer disease of Edward Jenner and, when aroused, the battle inspiration of Stonewall Jackson." In his time, he saw the mortality rate from tuberculosis drop from 190 per 100,000 population to 90, and typhoid from 33 per 100,000 population to five. He is commemorated in the Ennion G. Williams Hospital at the Medical College of Virginia, the cornerstone of which was laid by Governor Stanley in 1955 containing a photograph of Ennion G. Williams, copies of his certificates of appointments by successive governors, and other mementoes of his career. He had no ambition for personal gain or self glorification but remained a man of the people throughout his career. Had he been grasping or ambitious, he almost certainly could have achieved any position he desired in the United States in public health, but to do this he might have had to sacrifice some of his personal honor. On his death, an editorial in the *Richmond News Leader* was entitled "A Great Idealist Passes". His ideal of the provision of health services in every county and city of the State has borne fruition in the work of his successors and today, thanks to Dr. Ennion Gifford Williams, the State of Virginia has a Health Department comparable to any in the world.

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A Training Program *Malgre* Lui

BURKE M. SMITH, Ph.D.
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Although there is a need in the Commonwealth of Virginia for Clinical Psychologists with doctoral degrees, there is no educational program leading to such degrees.

ONE of the widely recognized needs of Virginia's total health program is for more and better training of professional personnel. This essay is a review of one aspect of one small segment of the larger need.

At the present time and in the foreseeable future there is no educational program in the State leading to a doctorate in Clinical Psychology, yet many positions for clinical psychologists in the State require a doctoral degree. As a consequence, all of the positions in the State occupied by doctoral level clinical psychologists are filled by persons who got their education in other states. (There may be a few exceptions who got their clinical training elsewhere and have taken a doctoral degree in a related field, such as education.) Recently the State Legislature enacted legislation which, in net effect, will ultimately make a doctoral degree mandatory for licensure either as a Psychologist or as a Clinical Psychologist. During the past decade, the recognition of the value of, and the demand for, psychological services has steadily increased, especially in the areas of mental health and hygiene.

It has proved difficult to induce many well qualified clinical psychologists to take positions in the state. There are a number of reasons for this difficulty, but the only one to be stressed here is that most psychologists want the opportunity to teach graduate psychology students as well as medical students, residents, nurses, teachers, and rehabilitation workers. He also wants a balanced program of teaching, research and clinical services.

In the past six years the Department of Neurology and Psychiatry at the University of Virginia School of Medicine has never had more than three doctoral level, faculty psychologists in the Clinical Psychology Section at one time, with usually only two. During these same six years, there have been 13 sub-doctoral (mostly Masters) clinical psychologists employed without faculty status, but never more than four at one time, and usually only two. Some of these persons have been able to work only part time.

These sub-doctoral psychologists have borne the brunt of the actual administration of psychological examinations but for reasons to be elaborated below, they have required a good deal of technical and administrative supervision from the faculty psychologists. This supervisory function erodes the time that might otherwise be spent in research and teaching non-psychologists.

The requests for psychological services are always in excess of our capability and it is necessary at times to limit quite arbitrarily the acceptance of referrals in order to maintain a reasonable balance of work load with available personnel. This arbitrariness has not always been fully appreciated by members of other departments in the school and hospital.

The kinds of patients, and the kinds of presenting problems, are quite varied, but

we rarely are asked to examine a patient with a simple or obvious problem. Usually there is a subtle diagnostic dilemma, a borderline question of competency, or a prognostic problem. Few psychologists are equally expert in all of the sub-specialties of the field, such as child psychology, adolescent psychology, neuropsychology, or gerontological psychology, yet we are constantly asked to see patients who fall in all of these categories. The problems created by some of these referrals for the masters level psychologists (as well as for the supervisors) can be appreciated in the light of the following paragraph.

The typical masters level psychologist that we have been able to employ is female, married or engaged to a student at the University (who is within one or two years of completing his education), and the proud holder of a brand new diploma. She may have had some practical clinical experience in the course of her training, but this is her first job. In addition to adapting to marriage and to a new community, she has the task of adapting to a demanding professional role. For at least one month, she is very unproductive but requires an inordinate amount of orientation and supervision. She has to begin to learn new psychological techniques and how to apply those in which she has had some training. She has to learn her way through a maze of hospital culture and tradition, and she has to learn to depend more on her own resources and devices. By the end of her fifth or sixth month she is quite productive but still needs, and gets, consultation on many of her cases. By the end of her ninth or twelfth month, when she is really capable, her husband will have graduated and she will have resigned to go wherever he has chosen to go.

It is this situation that is implied in the title of this essay. We have, in fact, been conducting a post-master's training program in spite of ourselves, but it has been a training program not recognized as such, not subsidized by any training grants, and from which neither the trainees nor instructors receive official recognition.

This is not to say that there have not been rewards to all parties. The hospital and patients have benefited from the services rendered, the psychology "trainee" has benefited from the accumulated training and experience so that wherever she goes she is better qualified to seek a new position, or to work for a higher degree. The supervisor also gains from this kind of experience, but relatively less so. He has the satisfaction of helping and watching the professional maturation of intelligent, strongly motivated young women. Also, in teaching, as should always be true, the teacher too learns.

Of the nine psychologists who have "graduated" from this program, all are employed in responsible positions as psychologists, but only one has remained in Virginia. Two of those who have left the state have earned doctorates, and one of the present employees will soon have completed his doctoral requirements. Two of the presently employed persons will be leaving their jobs and the state in the coming summer.

One of the inherent difficulties in such an informal training program is the problem of recruiting and maintaining a constant supply of replacements. For the most part we have had to wait for inquiries from wives or fiancees of incoming students or interns. We have never had a surfeit of qualified applicants and in some years we have been unable to fill our quota.

It would be helpful if this program could be formalized and given some financial support other than the present source, departmental earnings. Such support has not been available from NIMH because of the lack of affiliation with an approved doctoral program in psychology.

With a training grant it would be quite feasible to structure an internship program that would enable us to add didactic training to the present on-the-job training. It might also enable us to move towards some

kind of a degree or certificate granting program, ultimately even a doctoral degree.

The hazards of the latter proposal are rather foreboding, however. The policy of the American Psychological Association is to recognize and approve only those training programs set up within a university department of psychology. Even though our education and training program were second to none, lack of "official approval" would be a handicap, especially in inducing students to enter the program. Further, to provide an adequate number of various subspecialists to the faculty and adequate physical facilities (offices, laboratories, equipment) would not be easy.

It would apear that the optimal solution, optimal in terms of time, budget, facilities, and personnel, would be for some Virginia college or university to initiate a doctoral training program for psychology students who want to become clinical psychologists and for us to be affiliated with that school to provide an internship in which clinical experience and opportunities for research in the clinical field would be available.

There has been some recent interest shown in the latter proposition, but so far the only action has been some general recommendation from study committees.

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A Long-Term "Cold" and Coffee-Drinking

And then there was the 39-year-old waitress who complained for six months about her "cold". She had a low-grade fever almost every day. She suffered from chills, lack of sleep, and irritability. After losing 20 pounds, she weighed 107 when admitted to a Philadelphia hospital. Her trouble? Too much coffee-drinking. She drank 15 to 18 cups a day, the woman told her physician. Her cold symptoms disappeared after five days in a hospital, during which she was limited to one cup of coffee a day.

A report on this case of caffeinism appears in the December 18 Journal of the American Medical Association. The author is Hobart A. Reimann, M.D., of Hahnemann Medical College and Hospital, Philadelphia.

Several common household items can cause illness if used excessively and the illness can be mistaken for other disease.

A cup of coffee contains about one-tenth gram of caffeine. A single one-gram dose of caffeine causes mental confusion, shivering, tremor, vomiting, and diarrhea. Ten grams of caffeine is said to be fatal. The waitress's caffeine intake was probably more than 1½ grams a day. The intake was spread over several hours, however—probably the reason she was not more seriously ill.

Clinicopathological Conference . . . .

Relapsing Febrile Illness in a Child, with Hepatic Granuloma and Terminal Meningitis

Prepared and Edited by

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CLINICAL DISCUSSANT:

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This conference was held on September 30, 1967.

D.D. UVH # 58-35-35
Autopsy No. 11269

Clinical History

This 3-year-old girl was admitted in June, 1967, to another hospital because of fever of approximately eight days' duration. The child's mother had noticed that for the previous four weeks she had seemed less active than usual, had a persistent cough, and perhaps had intermittent low-grade fever. She had been given treatment at first for a presumed cystitis, and later a course of Erythromycin without effect. During the few days before the first hospitalization there had been high fever spikes in the afternoon. At this time it was learned that her birth and development had been unremarkable, including successfully treated iron-deficiency anemia at 18 months, and that she had a healthy twin and newborn sibling.

Physical examination at the referring hospital showed her to appear normally developed and nourished, afebrile, and non-toxic. The only abnormalities were enlarged liver and spleen. A few superficial ulcerations of the mouth grew out monilia. Laboratory studies included Hct 38%, WBC 3085, (56 segs, 35 lymphs, 2 monos, 2 eos). Cultures of urine, blood, bone marow and axillary node section were sterile. Chest x-ray showed a diffuse, fine infiltrate in both lungs. Tuberculin and histoplasmin skin tests were negative. Sputum for acid fast bacilli was negative. Initially the patient was given Triple Sulfa, and she was also given INH and streptomycin for 12 days. At one point she was also given chloramphenicol because of the possibility of pertussis. Initially there were high fever spikes, but after the first week there was disappearance of fever and symptomatic improvement. Mycostatin was given for oral moniliasis. Because of the negative studies for tuberculosis, INH and streptomycin were stopped after 12 days. Low-grade jaundice was present. Liver and axillary node biopsies were initially thought to show encapsulated yeasts, although this interpretation was later revised to indicate only granulomata composed of histiocytic cells with an occasional multi-nucleate giant cell. Clinical improvement accompanied the continuation of Triple Sulfa therapy, and she was discharged with a plan to continue that for two months. On 6/28—two weeks after admission—complement fixation tests for histoplasmosis were negative.

During the first two weeks at home she seemed relatively well, and then fever and cough re-appeared. During the few days prior to admission here there were anorexia, diffuse abdominal and extremity pain, and irritability. On the day of referral she had a generalized seizure.

Physical examination here showed an alternately restless and lethargic child, weight 15.9 Kg, T 38.4, P 140, BP 115/80. No dyspnea or cyanosis. There was neck stiffness and Kernig's sign was present. There was no lymphadenopathy, and the lungs sounded clear except for some increase in breath sounds at the bases. The liver was down 1 cm., the spleen 3.5 cm.

Laboratory studies here showed Hct 36%, WBC 7200 (16 bands, 60 segs), negative

tuberculin, blastomycin, histoplasmin and coccidiodin skin tests. Chest x-ray initially normal; skull x-ray normal. Spinal fluid was slightly xanthochromic, and contained: protein 258 mg%, glucose 56 mg%, 384 WBC (85% PMN, 15% lymphs), and 27 RBC. There were no organisms seen, and an India ink preparation was negative. Bone marrow showed non-diagnostic, increased histiocytes.

Hospital course here consisted of continuing lethargy and restlessness, progressing to semi-coma by the second hospital day. Ampicillin was started. Seizures recurred on 8/10/67, and there was fever of 40.6° C. INH and Amphotericin B were started. On 8/11/67 pupillary dilatation and inequality were present. An Ommaya reservoir was inserted through a left frontal burr hole; ventriculograms showed diffuse dilatation without obstruction in the ventricular system. Despite the above measures, including the intra-ventricular administration of Amphotericin, there was no improvement, and the child died at 2 A.M. on 8/14/67.

Clinical Discussion

Dr. Calvin Kunin: This is a 3-year-old girl, who was first admitted to another hospital in June, 1967, because of fever of eight days' duration. The illness may have begun earlier than that, on the basis of her mother's account of intermittent cough and low-grade fever for approximately three weeks. As often happens in situations which later become complicated, an apparently blind trial of an antibiotic—in this case erythromycin was given first without benefit. At the time of the first hospitalization it was learned that her birth and earlier development were unremarkable except for a slight iron-deficiency anemia. She was having high, spiking fevers, appeared relatively non-toxic, and the only abnormalities on physical examination were enlarged liver and spleen. It is interesting that her white cell count was below normal with a normal differential and a few eosinophiles. The two eosinophiles are important evidence against one of the major diagnostic possibilities—that is, in miliary or severe tuberculosis, eosinophiles are not seen. If tuberculosis is the correct diagnosis, I will have to assume that there was some technical error in the recording of those two cells. All of the appropriate bacteriologic studies were then performed—cultures of blood, sputum, bone marrow, and axillary node—and were negative. Skin tests were negative to both tuberculin and histoplasmin.

At this point the presence of granulomas in the liver was added to the picture of a febrile child with hepatosplenomegaly. I will come back later to the possibility of some identifiable organism being present in these granulomata. The next event in this story was the nearly simultaneous administration of two different antibiotic programs directed against the two likeliest causative organisms of such a picture. Actually there were three different programs given in a short time: chloramphenicol because of the possibility of pertussis, Triple Sulfa reportedly for histoplasmosis, and twelve days of INH and streptomycin for possible tuberculosis.

This girl was undoubtedly quite sick and febrile at her first admission, and the resorting to a therapeutic trial without a definite etiologic diagnosis might have been justified. It is too bad, however, that her doctors felt pushed into treating several different possibilities at the same time, because the interpretation of any apparent therapeutic response was obviously made uncertain. Also, I don't understand the use of Triple Sulfa for possible histoplasmosis. Current textbooks do mention some effectiveness of sulfonamides in animal infections with histoplasma, and possibly also in children, but Amphotericin is clearly the drug of choice. This would be especially true in disseminating infection with liver involvement. Her improvement while on Triple Sulfa seems to have been interpreted in the other hos-

pital as evidence in favor of histoplasmosis at that time. My interpretation would be that improvement under those circumstances would be more against histoplasmosis than in favor of it. Nevertheless, that diagnosis also received brief support from a preliminary report of encapsulated yeasts in the liver biopsy. This report was later revised to indicate only that granulomata with histiocytes and occasional multinucleate cells were present. By the time of the revised biopsy reading, however, the patient was doing better, and no evidence for tuberculosis had appeared. So treatment for tuberculosis was stopped, and she was sent home on sulfa.

Next this girl again developed fever and cough, and she came in with diffuse abdominal and extremity pain, irritability and a seizure. She again had enlargement of the liver and spleen, and now she had obvious meningitis. The white count was 7200 with 16 bands, 60 segs, and no eosinophiles. The spinal fluid contained high protein, normal glucose, and many white cells, 15% of which were lymphocytes. This indicates either a fungal or tuberculous meningitis, and is really a major tip-off in terms of the diagnosis. I am told that India ink preparations were done and were negative for cryptococci. She was transferred here in an advanced stage of illness, and by the second hospital day was in semicoma. Amphotericin was now given, and it was given by the most direct means possible—directly into the cerebral ventricles through an Ommaya reservoir. At this hospital we also seem to have felt the need for a shot-gun approach, since she was also given Ampicillin and antituberculous treatment. Despite these measures there was deterioration in terms of the central nervous system especially, and she died on the sixth hospital day. This would be a good point to look at the x-rays.

Dr. William Wilson: We were able to review a chest x-ray made at the time of this patient's first admission to the local hospital. This does show a fine, diffuse, soft nodular infiltrate in both lung fields. Either of the two major diagnoses which Dr. Kunin is considering would be likely causes of such a picture. Her chest x-ray on admission here was reported as normal, although that process could still be present. Later chest x-rays here show the development of an infiltrate in the apical segment of the left upper lobe. Since she had become comatose at that point, aspiration pneumonitis was a likely explanation. Another possibility for this sequence of changes in the chest might be infection with pneumocystis carinii.

The initial skull x-ray here was normal. The ventriculograms show dilatation of the lateral ventricles and to a lesser extent of the third and fourth ventricles. Air passed freely through the cisterna magna, showing normal midline structures. There is no evidence of a space-occupying lesion. Ventricular dilatation without obstruction, then, would probably add up to a communicating type of hydrocephalus.

Dr. Kunin: How do you explain such a hydrocephalus?

Dr. Wilson: Infections involving especially the base of the brain, such as tuberculosis tends to do, may cause this. Exudate presumably interferes with normal transfer of cerebrospinal fluid.

Dr. Kunin: It is always important to know something about the background of an individual, and this protocol has insufficient information. I would like to know her race and socio-economic level, and whether there might have been someone in the family with tuberculosis. That could have a real influence on the probability of that diagnosis here. Was the family tested for tuberculosis? That is the sort of information which I should have.

In any case, we are left to deal with a febrile illness, which lasted about three months, and which involved the lungs and liver and eventually the central nervous system. From the liver biopsy and spinal fluid findings I think we can limit the pos-

sibilities to infectious diseases. It is worth mentioning sarcoidosis, which would be the common cause of granulomas in the liver. Although sarcoidosis can involve the brain, it is unlikely to give you a febrile picture and meningitis as seen in this patient. I was intrigued by the possibility of *pneumocystis carinii*, and I wonder if Dr. Smith can tell us whether this is associated with granulomas in the liver.

Dr. David Smith: No, I don't believe it is.

Dr. Kunin: Good. We see pneumocystis infection almost entirely in individuals who have lupus or transplanted kidneys, in which large doses of steroids or other immunosuppressive drugs are being used. That would probably not be a consideration here.

To me the most important point in helping focus on the diagnosis here is the presence of granulomas in the liver. Dr. Smith was very kind to review the biopsy with me, and there was no question that granulomas were present. What causes granulomas? I have already mentioned sarcoid, and the reasons for considering that less likely in this case are the fever and meningitis. This leaves us with three other possibilities. One of these is fungi, specifically histoplasmosis. If you're lucky in histoplasmosis you can see a clear cell in the granulomas, filled with small, clear, PAS-positive bodies. These were carefully looked for and not found. Other fungi would have been recognized in a similar fashion if they had been present.

The most common cause of this combination of findings would be, I believe, tuberculosis. The people taking care of the patient also thought this, and she was given a short course of treatment for tuberculosis early in the illness. You can see, in retrospect, how the improvement of this child on her combination of antibiotic treatment could have been a trap. At this point her skin test and probably family history were negative, the acid-fast stains on all of the tissues obtained were negative, she was better, and treatment was stopped. This is one of the reasons why people talk about being "committed" to a given program of therapy. You do become committed because the treatment masks the events that you need to make the diagnosis more certain. This lulls you into confidence, and then really gets you in the end, if the treatment has been insufficient to completely eradicate the infection. There are times when a commitment can be broken—when all of the appropriate studies have been done and are reported negative. Perhaps this is what happened here, and tragically.

One possibility that did occur to me was toxoplasmosis, which can be characterized by pneumonitis, muscle tenderness, and central nervous system signs, especially in children, and is frequently missed. There are four clinical forms of toxoplasmosis. Most people are aware of the neonatal form with conspicuous retinal involvement and blindness. There are two forms with either encephalitis or generalized lymphadenopathy as the dominant aspect of a combination of those two features, with mononuclear cells in the blood and spinal fluid. There is also a form that is very much like typhus, including a generalized rash, but also having increased mononuclear cells in the blood, which was not the case. Fever is usually not marked. The importance of toxoplasmosis is that this is a disease for which we have therapy, and in which the organism can be identified. The diagnosis is usually made by the demonstration of toxoplasma antibody in serum with the Sabin-Feldman dye test as early as a week or so after infection, and the parasite may be recovered from mice which have been injected with blood or infected tissues. Asymptomatic, healed toxoplasmosis is a sufficiently prevalent condition that low antibody titers or positive skin tests are not necessarily meaningful. Actually, as I looked at this case with the negative fungal studies and negative tuberculosis studies and the central nervous system infection, my initial reaction was that this was, indeed, a case of toxoplasmosis. So I

asked Dr. Smith if there were any toxoplasma organisms, which look very much like histoplasma, in the liver biopsy, and he said, no. Furthermore, you don't see granulomas in toxoplasmosis, so that pretty well killed that diagnosis insofar as I was concerned.

Well, this brings us back again to tuberculosis because this is the common cause of granulomas that kill people. Now, why was the tuberculin test negative? One study of tuberculosis in childhood from Newcastle-on-Tyne reported that only 6.3% of their cases of tuberculous meningitis had a negative skin test to 10 units of tuberculin, and that they were nearly moribund at the time. This child was not that sick at the time of her first admission, so the literature leaves me little support for making such a diagnosis. Nevertheless, my effort to review the literature, although it did not help me explain the negative skin test, indirectly helped me in accepting tuberculosis as the diagnosis. I was so concerned about this being tuberculosis that I went to the library to get out Lincoln and Sewell's, *Tuberculosis in Children*, and it was checked out. Now, who was it signed out to? It was signed out to the pediatric resident who also was listed as being in charge of this case during her final, brief stay here. That may be a very clever trap, too, but somehow it seems to help my diagnosis.

So then we end up with three main possibilities—histoplasmosis, toxoplasmosis, and tuberculosis. It depends on how seriously you take the granulomas in the liver. I take them very seriously and base by diagnosis on this finding. It would be more likely to miss acid-fast organisms in these granulomas than to miss encapsulated yeasts, and I will choose the last of these three, that is tuberculosis. I completely sympathize with the position of the pediatricians in view of the negative tests, and the improving condition, but in retrospect with the presence of hepatic granulomas it was unfortunate to let this child go home after only an inadequate course of anti-tuberculous or antifungal therapy.

Dr. John Owen: I would take issue with your statement that sarcoid does not produce fever. I have seen high fever several times in sarcoid, and this is confirmed in a report in the Annals of Internal Medicine.[1]

Dr. Kunin: The progressive course with meningitis and neurologic involvement here was not like sarcoid.

Dr. Oscar Swineford: Why not torula?

Dr. Kunin: India ink preparations of the spinal fluid were negative. Incidentally, this would be an important differential diagnosis, it is being seen frequently, and is successfully treated with the same agent finally used here for histoplasmosis, namely Amphotericin.

Clinical Diagnosis

Subacute infection of the liver and meninges, probably due to histoplasmosis.

Dr. Kunin's Diagnosis:

Disseminated tuberculosis, with hepatic involvement and tuberculous meningitis.

Pathological Discussion

Dr. David E. Smith: Dr. Kunin has made reference to the liver biopsy which is illustrated in Figure 1. This shows a granuloma with a central multinucleated giant cell. The surrounding cells are histiocytes and lymphocytes, and in several places in the biopsy there were other granulomas composed only of such histiocytes and lymphocytes. Within the giant cell there are vacuoles which, in some instances, are obviously phagocytized lymphocytes, but in others have an appearance that was probably the basis of the suggestion that histoplasma might be present. Special stains have never shown an etiologic agent in these sections. This type of granuloma, with such distinct giant cells, rather limits the possible

suggestions of an etiologic agent. It is quite compatible with tuberculosis and also with histoplasmosis, as well as a few other fungi, such as coccidioidomycosis but is not to be expected in toxoplasmosis. It is compatible with sarcoid.

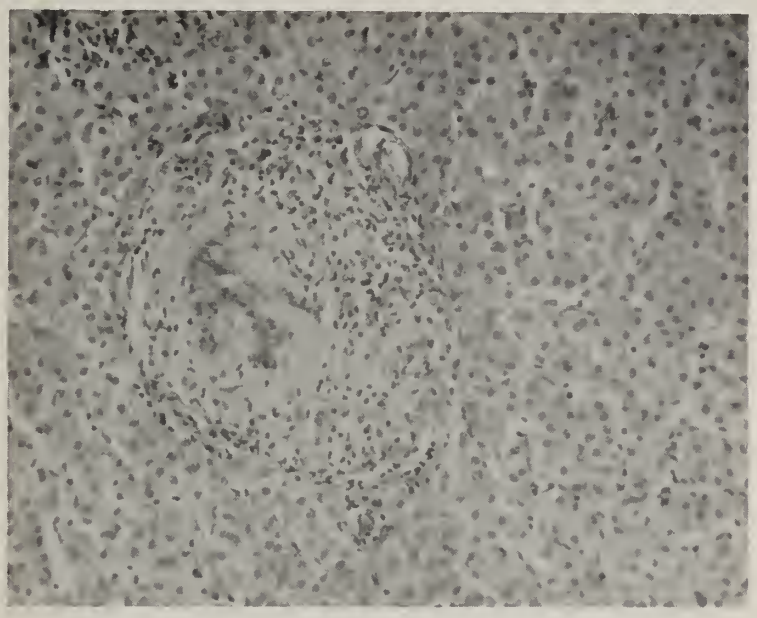


Fig. 1. Tubercle in biopsy of the liver. The giant cell is markedly vacuolated, but no etiologic agent was identified in this biopsy. (Hematoxylin and eosin stain; X 260.)

At the time of the autopsy, consequently, we were without definitive evidence as to what might be the etiologic agent. The first of the gross findings consisted of the recognition of miliary granulomas, less than 1 mm. in diameter, in the enlarged liver, the enlarged and hyperplastic spleen, and the kidney. It was therefore obvious that we were dealing with a disseminated infection that had probably spread by way of the blood stream.

The lungs were heavy, congested, and dark. The tracheobronchial and mediastinal lymph nodes were enlarged and hyperplastic, and several contained caseous nodules up to .5 cm. in diameter. In the upper lobe of the left lung there was a relatively well encapsulated, caseous lesion, almost 1 cm. in diameter. Combined with those nodules in the lymph nodes, this lesion made the characteristic Ghon complex. This complex is classically a sign of tuberculosis, but we have grown to recognize that it may be mimicked by other diseases, particularly histoplasmosis in this part of the country. Figure 2 illustrates the military infiltrates that were present throughout the lungs. Microscopically the caseous nodule in the upper lobe of the left lung was fairly well encapsulated but still showed signs of activity in



Fig. 2. Miliary tubercles in a lower lobe of the lung.

terms of the central necrosis, active histiocytes as well as fibroblasts, and the giant cells in the capsule. The lesions throughout the lung were collections of histiocytes and a few giant cells that obliterated the alveoli but had not progressed to much caseation. These foci were more apparently related to small blood vessels than they were to the bronchi, and are consequently to be considered miliary rather than bronchogenic.

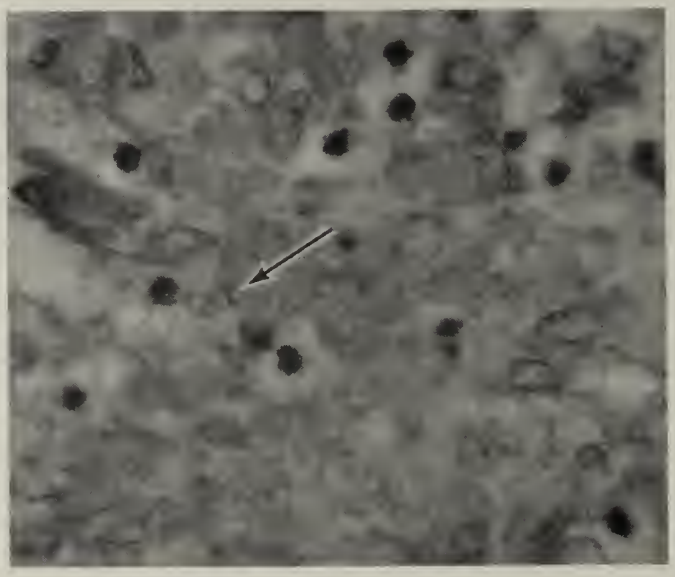


Fig. 3. An acid fast bacillus in a miliary tubercle in the lung. (Acid fast stain; X 1200.)

The etiologic culprit is shown in Figure 3. It is a rod-shaped acidfast bacillus that satisfies all the criteria for a tubercle bacillus. Bacteriologically, the culture is a little unusual in that the colonies are smooth, but I have refrained from referring to it as atypical because that adjective is presently used for other races that are characterized by chromogen production. This seems, instead, to be a relatively minor variant of the colony form of the tubercle bacillus.

The brain showed a rather thin and grossly difficult to recognize infiltration of the leptomeninges around the optic chiasm. About the medulla (Fig. 4) the exudate was thicker and a rather typical granulomatous or chronic meningitis. Figure 5, a histologic section in this area shows the tuberculous character of the meningitis with caseous necrosis, giant cells and histiocytes. This exudate had already involved blood vessels sufficiently that there were foci of ischemic necrosis in the brain substance.

We were not successful in demonstrating a tuberculoma that had ruptured into the subarachnoid space and initiated the meningitis, but our gross survey of this brain



Fig. 4. Cross section of the pons with thick granulomatous leptomeningitis.

was probably inadequate in that regard. The concept of the importance of a tuberculoma within the brain in the genesis of the subarachnoid infection is important, because it correlates well with the lag period between systemic evidence of the disseminated infection and the appearance of the signs of meningitis. Actually, in this case, it appears that the meningitis was apparent for only the last four days of the patient's life, the symptoms appearing shortly after admission to this hospital. By this time, probably a great deal of damage had already been done, and the process was irreversible.

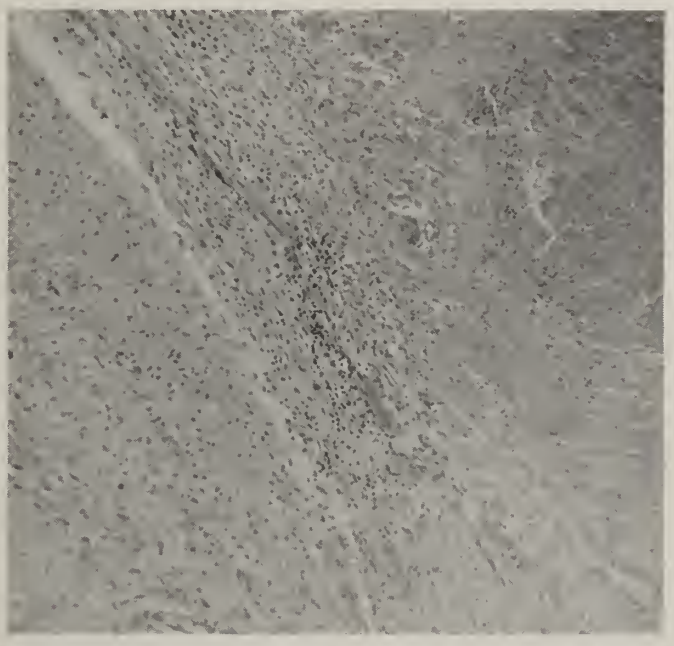


Fig. 5. Tuberculous meningitis about the medulla. Epithelioid cells, fibroblasts, giant cells, and caseous necrosis are prominent, and acid fast bacilli were demonstrated. (Hematoxylin and eosin stain; X 130.)

There is one aspect of the protocol on which I would like to comment. There was no description of the formation of a web on the cerebrospinal fluid specimen, although the protein was quite high. I suspect that we have presently grown out of the habit of watching spinal fluid for the formation of a web, but formerly it was considered a prime characteristic of tuberculous meningitis. Obviously it can form in other situations with high protein and fibrinogen contents, but it should always increase our suspicion of the diagnosis of tuberculous meningitis and lead to an especially thorough search for the bacilli. Such searches have extended for hours in times past to eventually be rewarded with success.

In retrospect, many features of this case seem to fit fairly well into place. The total duration, the presence of the pulmonary lesions, and finally the presence of the meningitis are fairly typical. The early therapy with INH and Streptomycin and the recounted improvement gives probably the best clinical indication that this was a case of tuberculosis.

Anatomical Diagnosis:

Tuberculous granulomas in the lung and mediastinal lymph nodes.
Miliary tuberculosis in the lungs, kidney, pancreas, thyroid, lymph nodes, liver, spleen and bone marrow.
Tuberculous meningitis.

Reference

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Cancer Trends . . . .

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Skin Cancer

JERRY W. BAINS, M.D.

Cancer of the skin is a disease which is potentially curable in almost 100 per cent of the instances. Unfortunately, delay on the part of the patients through ignorance or fear and errors on the part of the physician by a delay in diagnosis, improper initial treatment, and inadequate follow-up can result in situations which require multilating resections to cure the patient. Uncommonly, the patient may die as a result of extensive metastatic disease.

The malignant epitheliomas, basal and squamous cell carcinomas, are the most common malignant neoplasms affecting patients in this country. Epidemiologic studies have shown that the actinic rays of the sun play an important part in the etiology of the disease.[1] Those patients who have lightly pigmented skin and who are subjected to prolonged exposure to the sun over a period of years are particularly affected. Hair color is not always an important indication of the susceptibility to the disease; people who have blue eyes and black hair are often victims of skin cancer. Races with darker skin have less difficulty with skin cancer. In Honolulu, where there is a mixture of dark skin Orientals and light skin Caucasians, skin cancer is 45 times more common in Caucasians.[2] The disease is rare in Negroes; of 2,245 patients with skin cancer with records in the Tumor Registry at the University of Virginia Hospital, only 31 were Negroes. The older age group is more commonly affected since it takes many years of exposure to develop changes which cause malignant degeneration to skin cancer. In large series about 5 to 7 per cent of the patients are under 45 years of age.[1] Males are affected more than females by a ratio of about three to one. The exposed areas of the body such as the face, neck, ears, and dorsum of the hands are the primary sites nearly 95 per cent of the time.

The skin changes which precede the development of malignant epitheliomas should be recognized by everyone. The skin becomes ruddy, dry, and scaly. The most common premalignant lesion is a senile or solar keratosis. These small areas appear first as scaly pigmented, either flat or elevated, areas which may grow larger both in diameter and in height. Sometimes the scaling piles up so that cutaneous horns develop. These pigmented lesions usually degenerate into squamous cell carcinomas. The signs of malignant changes are inflammation, an increase in size, and ulceration. Bowen's disease is an unusual premalignant lesion which is more correctly termed a carcinoma in situ of the skin. This disease usually affects multiple sites and, strangely, in areas that are not exposed to the sun. Burn scars and other traumatic scars may undergo degeneration into epidermoid carcinomas; usually such scars are the sites of considerable trauma after healing. Although squamous cell carcinoma can develop surprisingly soon (within months) following healing of the wound, usually many years lapse before carcinomatous changes take place. Prior to the modern era of chemotherapy arsenical dermatoses were seen more frequently; these lesions, which seemed to have a propensity

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for the palms of the hands and the soles of the feet, often were beginnings of skin cancers. The long-term after effects of x-rays, especially from unfiltered sources, and the resultant malignant changes are well known to most physicians.

The two main types of malignant epitheliomas are basal cell and squamous cell carcinomas. Clinically, these are difficult to differentiate in the early stages but differentiation at this point is usually of little consequence. Basal cell carcinomas may present as small raised areas with no ulceration in the beginning. This nodular type is considered the least aggressive and most slowly growing. The ulcerating basal cell carcinoma (rodent ulcer) may begin as a nodular lesion early and then break down to an ulcerating lesion with rolled edges which spreads fairly rapidly over the surface of the skin destroying the skin and some of the subcutaneous tissue. These lesions may invade deeply and involve underlying bone and other vital structures. The cicatrizing basal cell carcinoma can be very deceptive. A flat plaque-like tumor which has irregular borders, it may invade the subcutaneous tissue more widely than can be appreciated by a palpation. Although basal cell carcinomas can develop in almost every site of the body, they are more likely to arise in areas of the skin containing abundant sebaceous glands. There is data which show that skin exposed to sunlight gives rise to basal cell carcinomas in direct proportion to the concentration of sebaceous glands therein. The chin, the cheek, the forehead are the areas that are most commonly involved. The area around the medial canthus of the eye demands especially early attention and treatment since deep invasion in this area can produce devastating results with loss of the eye or even invasion of the frontal and ethmoid sinuses to such an extent that resection for cure is not possible. Metastases from basal cell carcinoma are rare, so this disease kills by invasion of vital structures when neglected long enough or inadequately treated.

Squamous cell carcinoma usually arises initially as an ulcerating lesion and spreads more rapidly in most cases than basal cell carcinoma. Often the clinician must make the diagnosis on the basis of the rapidity of growth only. Approximately 5 to 10 per cent of the patients with squamous cell carcinoma of the skin will develop regional node metastases with a much smaller percentage of patients having distant spread by way of the blood stream. In the early stages, without evidences of lymph node metastases, local excision is always indicated. Of course, with nodal metastases regional lymphatic dissection is necessary.

The treatment of malignant epitheliomas of the skin may be by chemosurgical techniques (Mohs), irradiation, or surgical excision. The chemosurgical technique of excision of tumors is very successful if properly performed; there are too few people who are adequately trained in this technique to warrant endorsement at this stage. A variant of the Mohs chemosurgical technique is electrodesiccation with a needle and curettement. Dermatologists use this as the treatment of choice and have very good results with smaller lesions. Irradiation may be necessarily employed for people whose general condition will not allow extensive anesthesia and surgical excision. For smaller lesions, properly given x-ray treatment provides excellent cure rates. X-ray probably should not be employed as the treatment of choice on people who will return to dry climates or those who by occupational necessity must later be exposed to the sun's rays. Often times treated areas exposed to such conditions undergo radiation necrosis much earlier than expected. For this reason also this treatment should be reserved for the older age group. Lesions overlying cartilage close beneath the skin or in areas with little subcutaneous tissue such as the fingers or the dorsum of the hands should probably not receive x-ray therapy. Radiotherapy should not be employed for post-radiation recurrent or persistent tumors. Surgical excision, in the

opinion of a surgeon, is the treatment of choice for the malignant epitheliomas. Frozen section studies at the time of operation and permanent histologic studies postoperatively give indication of adequacy of excision. Late irradiation necrosis is not a problem, and properly planned and executed surgical treatment provides good cosmetic results.

Prophylactic measures should be advised for those patients who are lightly pigmented but, because of their occupation, are exposed to the sun's rays. Some indication of the necessity for prophylaxis may be obtained by the family history of the patient. If previous members of the family have been known to have skin cancer, probably the offspring and relatives of such patients should be advised to take prophylactic measures to prevent development of the disease. Adequate shading and covering of exposed skin with hats, gloves, and long sleeves are simple measures which can be instituted by anyone. Some of the newer sun screening lotions may provide an additional measure of prophylaxis.

The treatment of precancerous lesions is somewhat more difficult. Recent evidence has shown that dermabrasion of involved skin achieves a long-lasting improvement. Of course, such treatment should be undertaken only after the skin is carefully surveyed and all doubtful areas excised or at least biopsied. Dermabrasion may be done under local anesthesia and minimal hospitalization is necessary. There is general agreement that a bland lanolin-containing ointment may also inhibit development of the precancerous lesions. Such ointments should not be employed in an attempt to treat established skin cancer.

It is particularly tragic that patients are continually seen with advanced skin cancer which requires multilating surgical procedures to cure; occasionally patients are seen with such advanced disease that curative resection is not possible. This disease is preventable for the most part, easily detected, and curable in the vast majority of cases.

COMMENTS

The management of skin cancer, exclusive of melanoma, has been clearly outlined by the author from the thoughtful viewpoint of a surgeon. Its frequency, location and usually successful response to any one of several types of therapy unfortunately have led to some patients and physicians neglecting early and adequate treatment. In the majority of cases some form of surgical excision is probably the treatment of choice. This not only confirms the histologic nature of the lesion but also defines the adequacy of the margins of resection. Close followup is then essential to diagnose recurrence or new primary lesions in as early a stage as possible. It should be noted, however, that recent experience with local therapy utilizing 5-Fluorouracil in an ointment base has demonstrated unusual success in some types of skin cancer. This has not been covered in Dr. Bains' presentation but will be the subject of a subsequent article.

THE EDITORS

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Public Health . . . .

MACK I. SHANHOLTZ, M.D.
State Health Commissioner of Virginia

Radiation Safety and Control in Virginia: A Developing Program

Any significant exposure to radiation, whether from external sources, or from internally deposited radioactive materials has a deleterious effect on molecular structure when interactions occur between the radiation and constituent(s) of the molecule. On a gross scale such interactions result in observable tissue damage or other physiological manifestations. Statistically valid studies show that physiological changes may accrue to members of later generations due to damage to genetic material.

Radiation exposure, therefore, in any form and in any amount, is theoretically undesirable, but in medical diagnosis and therapy the choice becomes one of balancing benefit against risk.

The State Health Department assumes that when a physician prescribes radiation for any legitimate use he has decided that the benefit holds the major promise, and the risk, while not ignored, should be taken. This is in line with the Department's policy that at no time will it attempt to abridge or interfere with a duly licensed practitioner's prerogative to prescribe and administer doses, including radiation. In fact, the "Radiation Safety and Control Law" passed by the General Assembly in 1964 expressly forbids any interference of this nature.

The State Health Department does have the responsibility, however, of promulgating regulations to insure the safety and control of radiation producing equipment.

The State Health Department proposes to effect a comprehensive set of radiation-safety-and-control regulations generally commensurate with those of the U. S. Atomic Energy Commission. This broad regulatory concept is designed to culminate in a formal agreement with the State of Virginia and the U. S. Atomic Energy Commission which will result in State assumption of Atomic Energy Commission licensing and regulatory control over byproduct materials and other limited categories.

There are advantages when the State takes over, even though in a sense the State program becomes an extension of the Atomic Energy Commission. One advantage is proximity to the user which allows incomplete or ambiguous licensing and inspection information to be resolved promptly and adequately. Further, it permits greater aid to users in meeting licensing and regulatory requirements. When the regulations are completed they will be impartially enforced but with the intent of proffered assistance in solving problems associated with the program's major goal: radiological safety.

The old tried-and-true parameters of radiation protection remain as valid as ever: time; distance; shielding. Surveying and monitoring are certainly no less important. Judicious application of these tenets will pay tremendous dividends in reducing radiation exposures to acceptable limits. These principles and practices are those currently employed in safety activities of the State Health Department and will be the general basis for complying with maximum allowable exposure criteria incorporated in future regulations. It will be incumbent upon the user to provide whatever action is necessary to assure that under normal continuous operating conditions no person will be exposed to radiation or radioactive materials approaching the maximum allowable values. Such a program will widen the gap between the alternatives of "benefit" versus "risk" in favor of the benefits hoped for when radiation dosage is prescribed by the physician.

Mental Health . . .

HOBART G. HANSEN, M.D.

Patient Work Programs at Western State Hospital

After almost 100 years of abuse, work has been re-established in our State Hospitals as a reputable therapeutic service.

The following quotation from Superintendent Stribling's annual report of 1837 will help us to realize how important a role work once played at Western State Hospital: "This subject (work) is at present exciting a great degree of interest, in many parts of our country, and in the later reports of the Superintendents of various insane establishments, we find its importance advocated with a degree of zeal and earnestness, betokening the deep solicitude which is felt for its success. Already the legislatures in some of the states have rendered a prompt and liberal response to the calls made upon them for aid; and the institutions under their care, have been supplied not only with workshops and the various implements necessary in mechanical pursuits, but also, one with 20, another with 40, a third with 50, and fourth with 70 acres of land, in the cultivation of which the patients may be employed."

For almost one hundred years patients worked primarily to provide service to the hospital in the laundry, food service, buildings and grounds and on the farm. Eventually the hospital economy came to depend on this "institutional peonage". Work assignments were monotonous; neither offering stimulation or hope to the patient. At times assignments appeared to be antitherapeutic since they supported the patient's low self esteem. Occasionally the patient who performed best was maintained in a patient status so the hospital could continue to reap the fruits of his labor. For these reasons, virtually all patient work was abandoned after World War II and in the decade that followed.

Since 1962, Western State Hospital has explored the establishment of Industrial Therapy and Vocational Rehabilitation programs, and within the past year and a half, both have been initiated.

Three types of patient work programs now in existence at Western State Hospital will be reported on: Vocational Rehabilitation, CHIRP, and Work Therapy.

Vocational Rehabilitation

Our Vocational Rehabilitation program began in terms of patient participation on August 1, 1967. Fifty-two patients have been processed through and another 35 are presently involved in some phase of the program. These patients attend three work evaluation areas located within the Stribling Building. A fourth shop will be opened within the next few weeks.

Clients are housed in a two-ward building near the Stribling Building.

At present there are 28 members of the Vocational Rehabilitation staff. One Western State Hospital physician spends half his time as medical and psychiatric consultant. The Clinical Assistant Superintendent coordinates this program with other hospital activities and maintains its medical supervision.

Several patients have been referred to the Work Therapy and CHIRP programs for work tryout, evaluation, or work conditioning. There is also a close relationship between Occupational Therapy and Vocational Rehabilitation, and patients may be referred from O.T. to Vocational Rehabili-

HANSEN, HOBART G., M.D., *Superintendent of Western State Hospital, Staunton.*

Approved for publication by Commissioner, Department of Mental Hygiene and Hospitals.

tation through the patient's attending physician.

Since the Vocational Rehabilitation department is a new one, its relationship to other departments and to the community, and its potential in terms of case load and effectiveness, has not been fully demonstrated. However, so far, we have every reason to believe it will be effective in reducing the readmission rate as well as in helping patients to become self-supporting members of the community.

CHIRP (Community Hospital, Industry, Rehabilitation Program)

CHIRP at Western State Hospital was preceded in 1964 and 1965 by a sock-mending project. Although unsuccessful, the sock-mending project did point out very forcefully the need for a well-defined and organized program. We, therefore, took advantage of consultation with Mr. Earl Frost, Director of the CHIRP program at the Brockton V. A. Hospital. We also attended conferences on Industrial Therapy and received information from many other hospitals which were successfully using the CHIRP idea. Following this, in November of 1965, a meeting was held at Western State Hospital to inform key industrial leaders of the area of the proposed CHIRP program. Mr. Frost was the principal speaker and was joined by a panel of State leaders in Vocational Rehabilitation. Also attending were local State legislators and members of the press. The proposed CHIRP program was then presented to the State Hospital Board which approved it in principle.

In the spring of 1966, CHIRP was started. An attendant supervisor was the only staff member in the program at the time. Presently, three former attendants comprise the CHIRP staff.

There is an average of 25 patients in the program at any one time. These patients work under conditions almost totally identical to those found in industry and are paid a piece work rate, comparable to the hourly wage rate for employees in industry performing the same task.

During the last eight months of 1966 almost $4000 was earned by CHIRP patients. So far this year, patients have earned $3437.69 and it is estimated that total earnings for the year will approximate $5000. One patient has earned as much as $100 per 27-hour work-week.

The quality of work performed by patients within the CHIRP program has been exceedingly high. At the present time over 150 different operations are performed for Virginia Metalcrafters and not one item has been rejected. Compared to this, the rejection rate at Virginia Metalcrafters is understood to be about 20 per cent.

With practically no exceptions, patients improve markedly while participating in the CHIRP program. Socialization increases and the patient's appearance improves dramatically. Patients who have demonstrated little interest in anything beyond their delusional systems begin to relate in a natural way. The increase in self-confidence is apparent.

There is no patient in the program at the present time who was in the program when it started. One hundred and one patients have passed through the program and it would appear that a high number of these patients have remained out of the hospital, possibly because of the CHIRP work and social experience.

Some time ago, one of the patients involved in the CHIRP program had this to say about it: "It concentrates on social role as well as vocational role-social role in relation to family and community and his working situation in the community itself. You can help a patient abandon pathological behavior patterns for more normal ways of achieving satisfaction in many ways besides the use of intensive psychotherapy or even in group therapy. In the casual contacts of everyday life when there is a continuing concern shown for his dignity, where there is acceptance and appreciation, where there

is an attempt made to restore self-esteem and when the group becomes truly supportive. CHIRP could become a better laboratory in living."

As with the Vocational Rehabilitation program, we hope our CHIRP program can expand. However, at the present time the program is limited by the number of employees who can be spared. We also feel that it would be desirable to have employees in the CHIRP program identified by correct job title and description. This has been referred to the Department Personnel Director who is making an evaluation of the situation.

Work Therapy

Although big business, the Work Therapy program is totally operated by two former charge attendants.

It is in this program that we must be most careful to make a distinction between exploitation and rehabilitation. In order for a work therapy program to be therapy, assignments must be made for reasons of rehabilitation by prescription from the attending physician. It is important to have the cooperation of the employee work supervisor and to have a limit on the time a patient may work without clinical re-evaluation.

There are 88 patient work areas within Western State Hospital, but the majority of working patients work in laundry, housekeeping and food service. Some patients are paid, but an equal number work without financial remuneration.

Each one of the over 700 patient work assignments at Western State Hospital has been described. Working conditions, number of hours worked, and pay scale have been determined, and accurate records are kept of numbers of hours worked. In each case, the patient is referred to Work Therapy by prescription and the employee supervisor is required to make an evaluation every two or every four weeks.

There are also a limited number of patients who are employed in Staunton. In the past half year, the number has averaged about ten. In spite of the small number of community-employed patients, total earnings for the last available twelve-month period was $30,762.05.

Let's Reminisce!

THE VERDICT OF THE JURY—We have received a copy of a piece of music called the "Verdict March", composed by Eugene L. Blake. It can be played on either piano or organ. The title page is very handsome, containing portraits of Hon. Geo. B. Corkhill, Hon. J. K. Porter and Judge W. S. Cox; also a picture of the twelve jurymen who convicted the assassin of our late beloved President. Price, forty cents per copy, or three copies for one dollar. Postage stamps taken as currency. Address F. W. Helmick, Music Publisher, 180 Elm St., Cincinnati, O. (Virginia Medical Monthly, April, 1882)

Diagnostic Laboratory Medicine . . . .

H. M. SNELL, M.D.

Melioidosis

Melioidosis is a disease of man and animals caused by Pseudomonas pseudomallei, an aerobic, gram-negative, motile bacillus found endemically in Southeast Asia and first described by Whitmore in Rangoon, Burma, in 1911. Though since this time sufficient cases have been studied and reported to permit the disease to be well documented as a clinical entity, it has been a rare and seldom considered disease in the United States. All cases with exception of some occurring as laboratory infections have been in persons from the endemic area, chiefly Indo-China, the Malay States, Thailand, Ceylon and contiguous areas.

The presence of a large number of U.S. troops in Vietnam, in the endemic area, and the policy of annual rotation of these troops to stations in the continental United States make the possibility of physicians and laboratory personnel encountering the disease as a diagnostic problem less remote than formerly. Awareness of this problem will be essential in rapid and accurate clinical and laboratory evaluation.

The disease occurs in an acute, subacute and chronic form and may not be clinically evident until some time after an individual has left an endemic area. It may be protean in its manifestations, mimicing viral, rickettsial and fungal infections, all of which tend to make the diagnosis complicated.

The etiologic agent, *Pseudomonas pseudomallei,* has also been called *Pfeiferella whitmori, Malleomyces pseudomallei* and *Loeffflerella pseudomallei,* and has been isolated from surface waters and soils in endemic areas where it appears to exist as a saprophyte. There is no evidence to suggest that reservoirs need to exist in animals or man although rodents do have the disease. Cases have also been reported in pigs, sheep, cats and horses. The disease is not considered to be communicable from man to man, and infection is thought to occur as a result of ingestion of the organism or direct inoculation through broken skin. All age groups are susceptible, and serologic surveys in native populations in Southeast Asia show that as many as 15% have antibodies. Since the reported cases have either followed a fulminating septic course or a recognizable chronic one, the existence of these antibodies might suggest the occurrence of subclinical infections perhaps not unlike those of primary tuberculosis.

Early it was recognized that melioidosis was similar in its pathologic lesions and course to glanders, and further that *Pseudomonas pseudomallei* was similar to the causative agent of glanders, *Malleomyces mallei* (*Actinobacillus mallei*) though the two can be differentiated serologically and bacteriologically. As in glanders, melioidosis may be characterized by small pus-filled abscesses with so-called granulomatous margins occurring in skin or any organ system. Central nervous system abscesses have not been reported which is the one organ system excepted.

In the 300 or more cases reported since 1911, 90% have had the acute form characterized by sudden onset of high fever, shaking chills, prostration, vomiting and diarrhea. Septicemia occurs early and death may follow in 3-4 days. In the subacute form, disseminated abscesses (so-called granulomas) develop and may involve skin, lung, liver, spleen, bone, prostate and epididymis. In chronic cases draining sinuses may persist for years. Most commonly the disease presents as an acute pneumonia. Acute toxic pneumonitis, fulminating sep-

ticemia, asymptomatic pulmonary infiltration, localized extrapulmonary lesions, and asymptomatic individuals with positive serologic tests are all recognized as variants.

Laboratory findings in general are those of an acute bacterial infection with no single parameter except cultural isolation of the organism being diagnostic.

Since the organism grows well on ordinary culture media producing cream to yellow colonies 2 to 3 millimeters in diameter in 24-48 hours, the laboratory investigation of the disease in any individual with undiagnosed, toxic, pulmonary illness who has been in an endemic area should not be delayed. The organisms are bipolar staining gram-negative rods and can be differentiated from *Pseudomonas aeruginosa* by their inability to grow in 4% sodium chloride veal infusion agar. Agglutination, complement-fixation and hemagglutination tests may be of value in cases other than the acute form. Specimens for bacteriologic investigation may include purulent exudates, sputum, blood, urine and feces.

Sensitivity testing of the isolated organism is important in guiding the physician in the use of antibiotics, and should be continued at intervals throughout treatment to monitor the development of resistance which has been reported to occur under treatment. In the past, *Pseudomonas pseudomallei* has been found to be sensitive to chloramphenicol, tetracycline, kanamycin sulfate, novobiocin and sulfadiazine and resistant to streptomycin, cephalosporin, ampicillin, penicillin G and colistin.

With early recognition, subsequent laboratory isolation and antibiotic sensitivity evaluation, acute melioidosis with fulminating septicemia may have less than the dismal prognosis of 90-95% mortality.

Division of Clinical Pathology
Medical College of Virginia
Richmond, Virginia

A Year of Better Health Ahead

We can look forward to a year of better health in 1968, suggests the American Medical Association.

During the past year, medical progress was made which will help ensure that each of us has a longer, more comfortable life.

There were new developments in artificial and transplanted internal organs.

There was some success in vaccine development and hints of new biochemical weapons against viral diseases and continued follow-up of promising leads in the mystery of cancer.

An anti-measles campaign by the American Medical Association and the U.S. Public Health Service has been so successful that fewer measles cases were reported during the first half of 1967 than in any comparable period since measles record-keeping began in 1912.

Licensure of a mumps vaccine is expected in the near future. A vaccine against German measles (rubella) may be generally available within two years. A vaccine to prevent erythroblastosis fetalis (Rh disease) also may be licensed in 1968.

Unfortunately, there are health danger signals, too. The public health struggle with venereal disease is far from won—gonorrhea is more prevalent in the U.S. now than at any time in the last 20 years.

Heart disease, chronic non-tuberculous lung disease, alcoholism, and drug abuse continue to be outstanding U.S. public health problems. Cancer takes far too many lives.

As the AMA points out, we are nowhere near the end of the road to perfect health. Medicine makes no promises that we will ever get there—certainly not in the near future.



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study I[1]

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of the organisms were susceptible to oleandomycin*

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of the patients responded favorably to TAO® (triacetyloleandomycin)

*In some cases more than one pathogenic organism was isolated from the patient.

study II[2]

Effect of oral therapy with TAO, erythromycin, and cloxacillin on the survival time of Rhesus monkeys after intravenous inoculations of lethal doses of staphylococci, phage type 80/81. (8 monkeys in each group)

conclusion:

*Under the conditions of this study and the doses employed, it was found that

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was far superior to erythromycin, as was cloxacillin, a bactericidal agent, and of particular interest, "...bacteriostatic triacetyloleandomycin was as effective or perhaps superior to cloxacillin in preventing lethal (staphylococcal, phage type 80/81) infection."

*It should be pointed out that results obtained in an experimental study of this nature may not necessarily be directly extrapolated to the clinical situation as it pertains to man.

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INDICATIONS: Include streptococci, staphylococci, pneumococci and gonococci. Recommended for acute, severe infections where adequate sensitivity testing has demonstrated susceptibility to this antibiotic and resistance to less toxic agents. **CONTRAINDICATIONS AND PRECAUTIONS:** Not recommended for prophylaxis or in the treatment of infectious processes which may require more than ten days continuous therapy. In view of the possible hepatoxicity of this drug when therapy beyond ten days proves necessary, other less toxic agents should be used. If clinical judgement dictates continuation of therapy for longer periods, serial monitoring of liver profile is recommended, and the drug should be discontinued at the first evidence of any form of liver abnormality. When treating gonorrhea in which lesions of primary or secondary syphilis are suspected, proper diagnostic procedures, including darkfield examinations, should be followed. In other cases in which concomitant syphilis is suspected, monthly serological tests should be made for at least four months. Contraindicated in pre-existing liver disease or dysfunction, and in individuals hypersensitive to the drug. Although reactions of an allergic nature are infrequent and seldom severe, those of the anaphylactoid type have occurred on rare occasions. When used in streptococcal infections, therapy should be continued for ten days to prevent the development of rheumatic fever or glomerulonephritis. The use of antibiotics may occasionally permit overgrowth of nonsusceptible organisms. A resistant infection or superinfection requires re-evaluation of the patient's therapy. In the event such occurs with this drug the medication should be discontinued, and specific antibacterial and supportive therapy instituted.

References: 1. Isenberg, H. D.: Clinical Evaluation of Laboratory Guidance to Antibiotic Therapy; Health Laboratory Science (July) 1967. 2. Saslaw, S., Carlisle, H. N.: Studies on Therapy of Staphylococcal Infections in Monkeys. 1. Comparison of Cloxacillin, Triacetyloleandomycin and Erythromycin. Proc. Soc. Exp. Biol. & Med.: Vol. 125, No. 4 (Aug.-Sept.) 1967.

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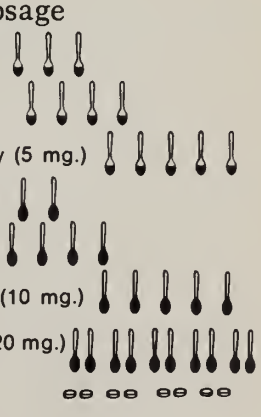
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Side Effects: Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness, insomnia, numbness of the extremities, headache, blurring of vision, swelling of the gums, euphoria, depression and general malaise.

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3-6 mo. . .½ tsp.* t.i.d. (3 mg.)
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5-8 yr. . . .1 tsp. q.i.d. (8 mg.)
8-12 yr. . .1 tsp. 5 times daily (10 mg.)
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or 2 tablets q.i.d.



*Based on 4 cc. per teaspoonful.

Maintenance dosage may be as low as one-fourth the initial daily dosage.

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When the agitated businessman goes to work...

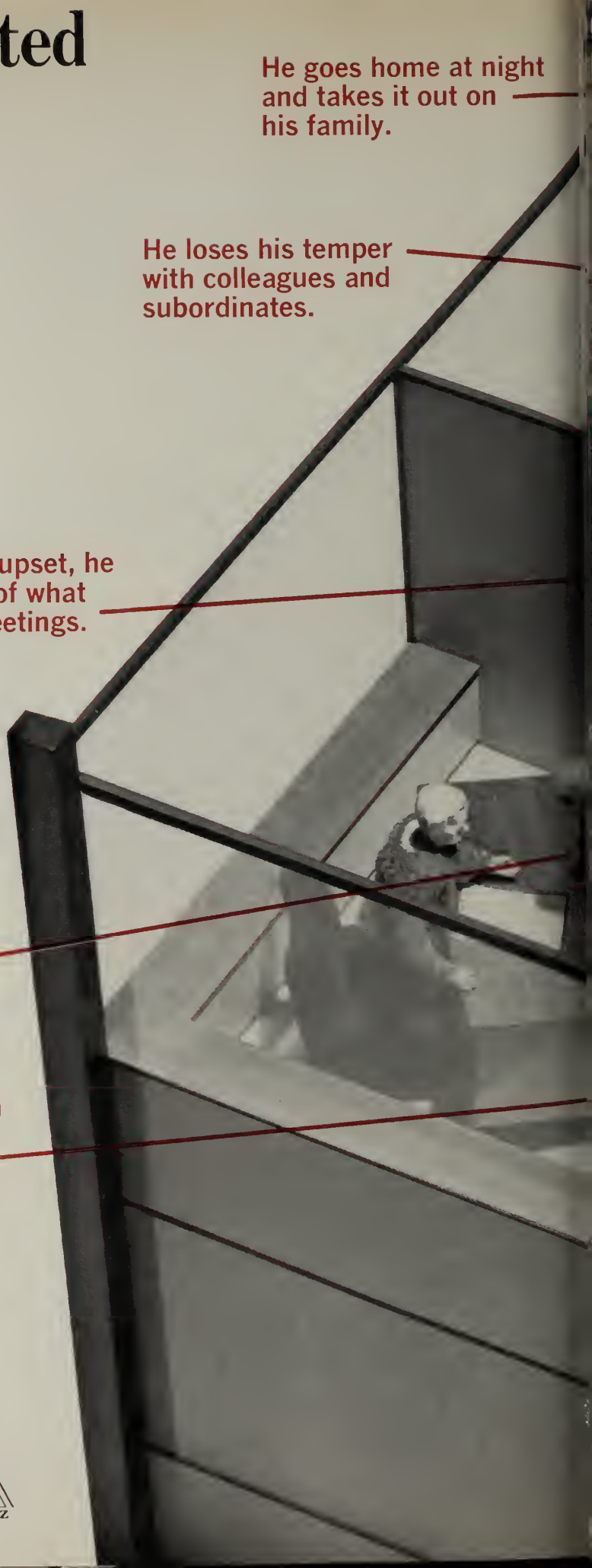
He goes home at night and takes it out on his family.

He loses his temper with colleagues and subordinates.

Emotionally upset, he misses half of what is said at meetings.

He is always fearful about his standing with the boss.

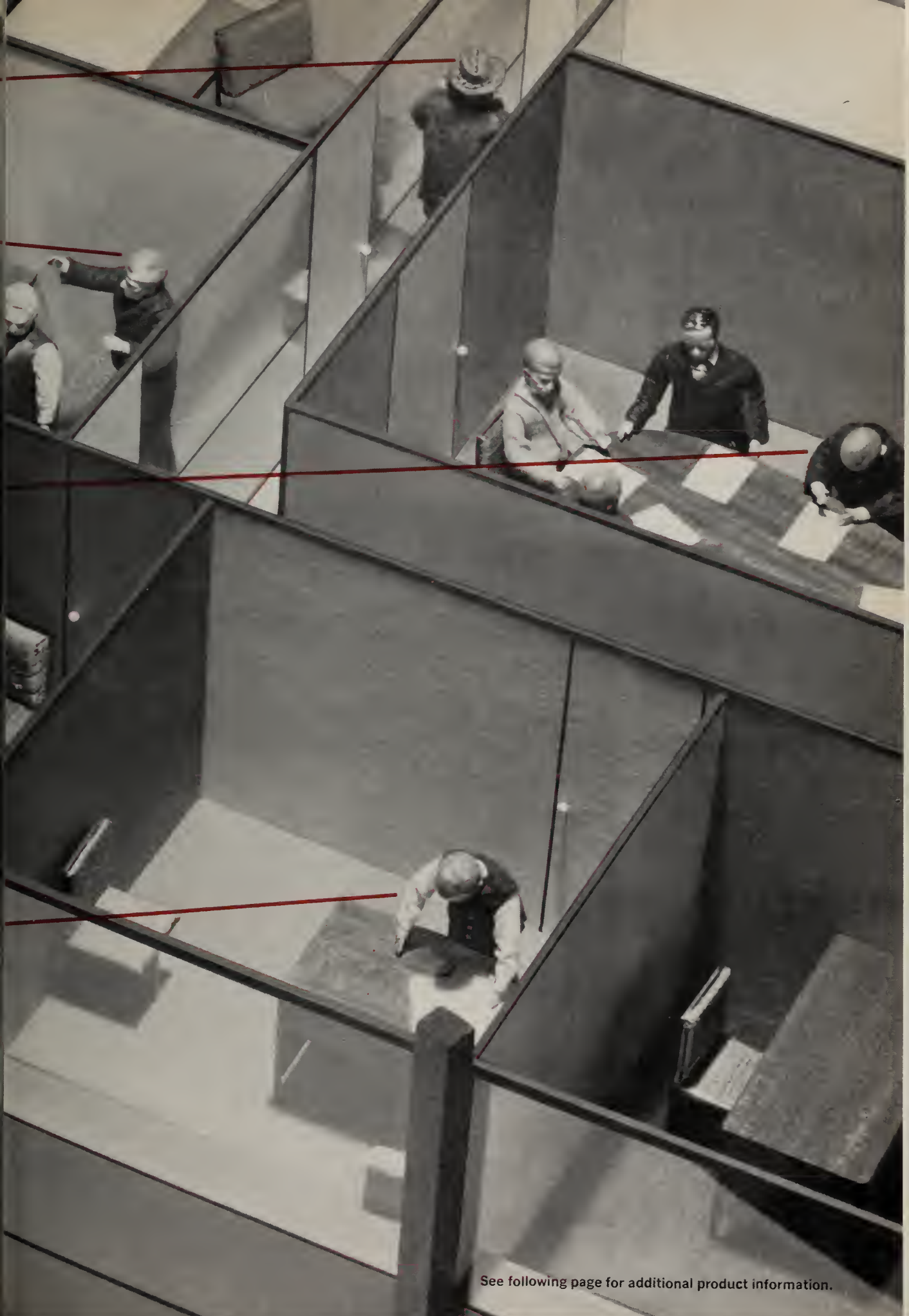
He just can't seem to settle down to his work.



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Mellaril®
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25 mg. t.i.d.

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Anxiety that *seriously interferes* with the individual's performance at work, at home, or in the community may be regarded as *moderate to severe* in degree.

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- often alleviates anxiety-induced somatic complaints
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Thus, when you consider the anxiety moderate to severe... consider Mellaril.

Contraindications: Severely depressed or comatose states from any cause, and in association with or following MAO inhibitors; severe hypertensive or hypotensive heart disease.

Precautions: Hypersensitivity reactions (e.g., leukopenia, agranulocytosis) and convulsive seizures are infrequent. Pigmentary retinopathy has been observed where doses in excess of those recommended were used for long periods of time. May potentiate central nervous system depressants, atropine, and phosphorus insecticides. Where complete mental alertness is required, administer the drug cautiously and increase dosage gradually. In addition, orthostatic hypotension (especially in female patients) has been observed. Epinephrine should be avoided in treatment of drug-induced hypotension.

Side Effects: Pseudoparkinsonism and other extrapyramidal disorders are infrequent; drowsiness, especially in high doses early in treatment, may occur; nocturnal confusion, dryness of the mouth, nasal stuffiness, headache, peripheral edema, lactation, galactorrhea, and inhibition of ejaculation are noted on occasion; photosensitivity and other allergic skin reactions may occur but are extremely rare.

Before prescribing, see package insert for full product information.

for moderate to severe anxiety

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Each tablet contains:

Potassium Iodide	195 mg.
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Ephedrine HCl	16 mg.

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Precautions: Usual for aminophylline-ephedrine-phenobarbital. Iodides may cause nausea, long use may cause goiter. Discontinue if symptoms of iodism develop.

Iodide contraindications: tuberculosis, pregnancy.

DOSAGE

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Woman's Auxiliary....

Bargain Prices for Doctors' Wives at VAMPAC—For $15.00 instead of the regular $25.00, doctors' wives may become members of VAMPAC—Virginia Medical Political Action Committee. This also includes membership in AMPAC. To get this very special price, our President, Mrs. Daniel Anderson, has promised that we would triple our membership. Let's get busy and send our checks TODAY to: Dr. James M. Moss, 3805 Florence Drive, Alexandria, Virginia 22305. Be sure that your husband is a member too. Remember that as members of the Auxiliary, we have an obligation to be politically active. We *must* support candidates whose philosophy is akin to that of the medical profession. Now, as never before, it is urgent for us to elect candidates in both state and federal governments whose voting record and political principles show them to be friends of medicine. In 1966, all of the candidates supported by VAMPAC emerged victorious. Let your check help insure the success of medical political action by joining the VAMPAC team in 1968.

How About This for a "Treat Instead of a Treatment"—One of the most interesting and exciting projects around these days is that of The Auxiliary to the Medical Society of Virginia Beach. This group has set up and is operating an arts and crafts therapy room in the Annex of the General Hospital of Virginia Beach. This is primarily for the emotionally ill and convalescent patients who are able to work with arts and crafts, such as knitting, ceramics, paper flowers, etc. They are fortunate to have a kiln for the ceramics, which was donated to the Virginia Beach Hospital by Dr. H. Lee Chandler, Jr., of Norfolk. Other groups have assisted in this project. The Princess Anne Woman's Club has made a generous donation and one of their members, Mrs. P. C. Hartman, comes one day a week to teach ceramics. The room is staffed by members of the Hospital Auxiliary. The Medical Staff have been most enthusiastic and their wives have felt it a most rewarding experience. If you are interested in getting such a project underway in your area and would like more information, write to Mrs. R. N. Redford, at 1325 North Bay Shore Drive, Virginia Beach, Virginia 23451.

WASAMA—and $5.00—Mrs. William Reardon has reported that the Fairfax Auxiliary has become the first sustaining member of WASAMA (Woman's Auxiliary to the Student American Medical Association). Mrs. Pinson Neal, State Chairman, is mailing forms to the Auxiliaries and urges each to donate $5.00—the amount of a sustaining membership. These funds are used to finance the Annual Conference of WASAMA officers and is so important as it is the only way that WASAMA can function. Let's not let them down!

While You Have Your Check Book in Hand—Remember A.M.A.-E.R.F.—Every day—every week—every year the medical colleges are faced with the problem of constantly rising costs. It is the purpose of this program to provide the deans of medical schools with an unrestricted source of funds. Also the Loan Guarantee Program assists in financing a medical education without a subsidy of any kind. It may be used by any student, intern or resident in full-time training at an approved medical school or hospital. And raising funds for the Institute for Biomedical Research, supported solely by A.M.A.-E.R.F., is the newest Auxiliary project. Your contributions will provide funds to attract the highest caliber of scientists and technical assistants. May your enthusiasm continue. Please encourage others to join in maintaining the high standards of medical education in America. Will you urge your husbands and medical societies

to make their checks to the A.M.A.-E.R.F. Auxiliary Fund—to help our State Auxiliary meet our goal or better yet—go over the top? Checks should be mailed to: Mrs. W. C. Fitzgerald, 172 Westminster Court, Danville, Virginia 24541.

Health Careers—Mrs. Carl Stark, State Health Careers Chairman, recently attended the Regional Auxiliary Workshop in Philadelphia and reports that the importance of Health Careers Chairmen was most strongly felt. The necessity of helping to solve the shortage of medical and paramedical personnel is certainly one of the prime objectives of all auxiliary members, for without adequate personnel in these fields, the future of medicine and the general welfare of our country will most certainly suffer. She stressed that auxiliaries must help by *recalling* inactive nurses back to work, by *reviewing* the overall state situation pertaining to health manpower and determining more effective and efficient ways to utilize existing health manpower, and by *recruiting* people into various health professions. In order to effect such a program, she strongly urges auxiliaries to first become well-informed by presenting the Health Careers Packet Program to their groups. One packet is allotted to each Auxiliary and may be obtained by writing to Woman's Auxiliary to the A.M.A., 535 N. Dearborn Street, Chicago, Illinois 60610.

When ordering, give the name of your auxiliary and the name and address of the person to whom it should be sent. It is hoped that you will become interested in the critical health manpower shortage, and will promote a Health Careers Day in your community this Spring. Mrs. Stark will be more than happy to assist you in any way.

Vision Problems after VD Treatment

Vision problems are showing up in a scattering of persons who received penicillin treatment for venereal disease several years ago and were thought to be cured, the Journal of the American Medical Association reported January 1st.

The report in the Medical News section of the Journal cautions that (1) the suspect organism in victims' eyes hasn't definitely been identified as related to venereal disease, and (2) investigators aren't certain that the organism itself causes the sight problems, which include blurring and partial loss of vision. Nevertheless, the findings pose serious questions about the treatment of venereal disease.

Further investigation is urgently needed, said Leslie C. Norins, M.D., Ph.D., director of the U.S. Public Health Service's Venereal Disease Research Laboratory at Atlanta.

Finding of the organisms in the eye was first reported by J. Lawton Smith, M.D., of the University of Miami School of Medicine.

Dr. Smith and his co-workers have found an organism in the eyes of patients which resembles T. pallidum. Other investigators question, however, whether this is its correct identification. Dr. Smith told JAMA Medical News that he and his co-workers have found the organism in the eyes and at 11 other body sites of 100 patients. At least 30 of these persons were known to have received "adequate penicillin therapy" and in a few cases, massive doses of penicillin for syphilis. Most of the 100 patients have a history of congenital or acquired syphilis.

None had any sign of active disease at the time they were examined—except eye and nerve lesions often associated with late syphilis.

Editorial . . . .

Southern Living and Other Things

HENRY W. GRADY, who for many years was editor of the *Atlanta Constitution,* when this was a conservative newspaper, once described a funeral in Pickens County, Georgia, which he said was "peculiarly sad". It seems the deceased had been a man of limited means who lived and died in a state which was still impoverished as an aftermath of the Civil War. He was buried in the midst of Georgia marble quarries yet the little tombstone they put above him came from Vermont. His grave was surrounded by a pine forest but the pine coffin was imported from Cincinnati. He had lived only a stone's throw from an iron mine, yet the nails in his coffin and the iron in the shovel that dug his grave came from Pittsburgh. He was buried in a New York coat and a pair of Boston shoes. His trousers were made in Chicago and his shirt came from Cincinnati. Grady opined that the South didn't furnish a thing on earth for that funeral but the corpse and the hole in the ground.

This was not an isolated example. The North has always felt that such a relationship should exist with the South. The North, in fact, had shown an earnest desire to bury the South long before either Grady or Khrushchev was born. This has been especially so in the publishing world. If we buy a national magazine or a novel we almost always find it was published in the North. If we look further we will find that if any reference is made to the South, it is usually in a derogatory way. The publishers and editors are not interested in anything favorable about the South and the writers found out long ago that a manuscript or news item must be slanted in an unflattering light if they wish it to be accepted.

Life, Time and *Look* are the worst offenders and the *Saturday Evening Post* tries to keep up with the others in its own inadequate way. The one outstanding exception, the *U. S. News and World Report,* is edited by David Lawrence, who does not have any axe to grind. The venom displayed by our critics often borders on the psychopathic. The late Thomas Lomax Hunter, who wrote a column in the *Richmond Times Dispatch,* frequently pointed out that people hate up and never down. He felt that an element of envy was a necessary component in order to promote a good hate. This may be the answer. Perhaps our critics sense that an indefinable something in our way of life has escaped them and they resent it.

Be that as it may, we need a forum to present what we have to offer. Recently a magazine designed to fill this void appeared, with the happy title of *Southern Living.* It is published by *The Progressive Farmer* of Birmingham, Alabama, an agricultural journal of considerable age and wide circulation. *Southern Living* has just completed its second year and

its subscribers have passed the half million mark. Its circulation is increasing at the rate of 100,000 per month.

This magazine deals with various aspects of life below the Potomac and Ohio rivers with especial emphasis on the amenities peculiar to this region. In addition to full length articles there are a number of regular features designed to make our critics even more envious of us. The current monthly average temperature and precipitation in various southern cities must have an added appeal in the snowbound North. Recreational spots and sporting events for each season are listed. The December issue included activities in Alexandria, Hampton, Williamsburg, Charlottesville and Richmond. Foods and cooking peculiar to the South and architectural aids and home adaptations are regular features. Gardening and landscaping in a southern climate are described during each appropriate month. Some of the information and monthly listings described here may appear dull but this is not the case and the rapid growth of this well-balanced magazine bears out the breadth of its appeal. *Southern Living* manifests the good manners we like to associate with this region by making no references to other more benighted parts of this country. There is no mention of the riot-torn cities to the North or to the lawlessness or labor strife in those areas. If we have the answers to some of the questions that vex the North, and apparently we do, for we have been singularly free from such problems, *Southern Living* has thus far played the role of the good neighbor and has not forced unsolicited advice upon the less fortunate. If this one lesson could be brought home to *Time*, *Life* and *Look* the thought and effort that have gone into this publication would be worth many times over.

The North, in fact, is completely ignored. Perhaps there is an indirect reference to other regions, for the subscription rate is only three dollars per year to us whereas the cost rises to four dollars when it crosses the Potomac. Most of the members of The Medical Society of Virginia will enjoy reading this magazine and having it in their waiting rooms. It is suggested that our members who practice in Washington should have *Southern Living* sent to their homes in Virginia rather than to their offices for otherwise it will be more expensive and there is always the possibility that this magazine may be barred from the mails if the comparison between the two sections becomes more evident.

H. J. W.

News....

Calendar of Events

Stoneburner Lecture Series on Nephrology—Medical College of Virginia—Richmond—February 22-23, 1968.

Virginia Chapter, American Academy of Pediatrics and the Virginia Pediatric Society—Annual Meeting—Williamsburg Inn and Lodge—Williamsburg—February 23-24, 1968.

Nuclear Medicine for the Clinician—Presented by Medical College of Virginia School of Medicine—Williamsburg—February 27-March 2, 1968.

Head and Neck Anatomy—A Post-Graduate Course of the Medical College of Virginia—Richmond—March 5-8, 1968.

AMPAC National Workshop—Sheraton-Park Hotel—Washington, D. C.—March 9-10, 1968.

Sectional Meeting for Physicians and Nurses—Sponsored by American College of Surgeons—Williamsburg—March 11-13, 1968.

Clinical Cardiology—19th Annual Post-Graduate Day Program of Roanoke Memorial Hospital—Roanoke—March 21-22, 1968.

Second National Congress on Socio-Economics of Health Care—The Palmer House—Chicago, Illinois—March 22-23, 1968.

Southeastern Surgical Congress—Annual Meeting—Sheraton Park Hotel—Washington, D.C.—March 25-28, 1968.

Newer Aspects of Cardiac Therapy—Symposium presented by Roanoke Valley Heart Association—Hotel Roanoke—Roanoke—2:00 p.m.-5:00 p.m., March 28, 1968.

Second National Congress on Medical Ethics—Drake Hotel—Chicago, Illinois—March 30-31, 1968.

Annual Clinical Conference—Louise Obici Memorial Hospital—Suffolk—April 3, 1968.

Annual Joint Cardiac Symposium—Sponsored by Heart Association of Northern Virginia and Washington Heart Association—Marriott Twin Bridges Motor Hotel—Arlington—April 10, 1968.

Cardiovascular Responses to Anesthesia—Fifth Annual Spring Symposium of Virginia Society of Anesthesiologists—Sheraton Motor Inn—Richmond—April 19-21, 1968.

The Child With Heart Disease—Recognition and Management—Continuing Education Program of the Medical College of Virginia—Richmond—May 9-10, 1968.

Virginia Academy of General Practice—Annual Scientific Assembly—The Chamberlin—Fort Monroe—May 9-12, 1968.

Seaboard Medical Association—Annual Meeting—Nags Head, North Carolina—June 21-23, 1968.

9th Annual Cardiovascular Symposium—Sponsored by Tidewater Heart Association and Council on Clinical Cardiology, American Heart Association—Lake Wright Motel—Virginia Beach—September 6-7, 1968.

New Members.

The following new members were received into membership of The Medical Society of Virginia during the month of December:

Thomas Herbert Bain, M.D., Crozet
Alan Seth Deutsch, M.D., Alexandria
William O. Harris, Jr., M.D., Richmond
James Lawrence Lynde, M.D., Lynchburg
Norman Bryant Thomson, Jr., M.D., Norfolk
Arthur Alvin Turner, M.D., Newport News
Charles Florian Woodhouse, M.D., Clifton Forge

Richmond Academy of Medicine.

Dr. Earnest B. Carpenter has been installed as president of the Academy, succeeding Dr. William H. Higgins, Jr. Other officers are: Dr. Richard A. Michaux, president-elect; Drs. W. H. Harris, Jr., and Ernest P. Buxton, Jr., vice-presidents; Dr. Richard W. Dodd, recording secretary; and Dr. H. Hudnall Ware, III, sergeant-at-arms.

Smith County Medical Society.

Dr. George E. Walker, Marion, has been elected president of this Society, succeeding Dr. O. O. Smith, also of Marion. Dr. E. B. J. Whitmore, Marion, is vice-president, and Dr. Henry R. Miller, Saltville, secretary-treasurer.

Dr. Richard E. Palmer,

Alexandria, has been appointed to the Committee on Welfare Services of the American Medical Association Council on Medical Service.

Dr. Mason C. Andrews

Has been selected to receive the Cosmopolitan Club's distinguished service medal as Norfolk's "first citizen" in 1967. Formal recognition of Dr. Andrews was made at the banquet on January 25th.

Dr. William Nash Thompson

Has been presented a pin for 24 years of perfect attendance by the Stuart Rotary Club. He is a charter member and past president of this Club.

Dr. W. Conrad Stone,

Roanoke, has returned to South Viet Nam to begin a second two-month tour in an ophthalmology program against eye disease. He was the first volunteer to start organizing this program in October 1966. The need for major eye treatment had been underscored by a U. S. military medical team that discovered extensive eye disease in the Delta. The program was started at Vinh Long Provincial Hospital in the Delta south of Saigon. The long-range goal is the establishment of a full-fledged eye center for South Viet Nam.

Dr. J. Edwin Wood, III,

Charlottesville, has been elected chairman of the Council on Circulation of the American Heart Association. As chairman he will represent the Council on the Board of Directors and Executive Committee of the Association and its Central Committee for Medical and Community Program. Dr. Wood is professor of cardiovascular research at the University of Virginia School of Medicine.

Section Officers of Southern Medical Association.

At the annual meeting of the Southern Medical Association in Miami Beach, November 13-16, the following Virginia physicians were named officers in various sections:

Dr. John Paul Jones, Richmond, vice-chairman of the Section on Pediatrics.

Dr. M. Pinson Neal, Richmond, chairman-elect of the Section on Radiology.

Dr. John T. Farrar, Richmond, secretary of the Section on Gastroenterology.

Dr. William P. McGuire, Winchester, chairman of the Section on Ophthalmology.

Dr. Claude C. Coleman, Jr.,

Richmond, was a guest speaker for the Third Annual Symposium on Tumors of the Head and Neck, sponsored by the Surgical Staff of the Pennsylvania Hospital, Philadelphia. His topic was Experiences in the Treatment of Far Advanced Cancer of the Mouth and Pharynx.

Dr. Jantz Honored.

The Bedford Lions Club, at its annual football banquet, honored Dr. J. G. Jantz for thirty years service as volunteer physician and surgeon to the athletic teams of Bedford and Liberty High Schools.

Hospital Medical Staffs.

Dr. George T. Hocker, Leesburg, has been elected chief of the staff of the Loudoun Memorial Hospital. Dr. John D. Wynkoop, Leesburg, is vice chief and Dr. Michael M. Vlahos, Sterling Park, secretary-treasurer.

Dr. John B. Sherry has been named president of the medical staff of the Waynesboro Community Hospital. Dr. DuBose Eggleston is vice-president, Dr. John P. Heatwole, treasurer, and Dr. William K. Orr, secretary.

Dr. Margaret D. Gleason

Has been appointed to the psychiatric staff of the Veterans Hospital in Salem.

Location Wanted.

Anesthesiologist, FACA, wanted relocation to State of Virginia. Tidewater area preferred. Licensed, experienced, fee for service preferred, group or partnership accepted. Write #135, care Virginia Medical Monthly, 4205 Dover Road, Richmond, Virginia 23221. (*Adv.*)

Hospital Physicians Wanted.

The Medical Staff, Board of Directors, and Administration of Winchester Memorial Hospital, Winchester, Virginia, are agreed to a plan of four full time physicians to be in private general practice limited strictly to the Emergency Department of the Hospital. Average work week is forty-two hours. A generous guarantee has been established.

Four experienced physicians having a Virginia license are needed. The target date is July 1, 1968. Interested physicians contact the Committee Chairman, James A. Miller, M.D., Box 554, Winchester, Virginia. Telephone 662-0377 (area code 703). (*Adv.*)

Wanted.

Qualified anesthesiologist to head department at C & O Hospital, Clifton Forge, Virginia. Income in excess of $30,000, largely on fee for service basis. Apply to J. M. Emmett, M.D. (*Adv.*)

Obituary . . . .

Dr. Claude Marshall Lee,

Warsaw, died December 20th at the age of eighty-five. He was a native of Fauquier County and a graduate of the School of Medicine of the University of Virginia in 1905. Dr. Lee served for forty-two years as a medical missionary of the Protestant Episcopal Church in Wusih, Kiangsu, China. He was founder and director of St. Andrew's Hospital in that city. During World War I, Dr. Lee was a medical officer with the rank of Major and served in Siberia. In World War II, he was made prisoner by the Japanese Army in China, but was exchanged and returned to the United States on the exchange ship Gripsholm. After the war, he returned to his work in China until shortly before that country came under the control of the Communist regime. During this post-war interval, he was made an Honorary Citizen of the Republic of China, a rare distinction known to have been accorded to only two other Americans. For many years, Dr. Lee was secretary of the China Section of the American College of Surgeons. He had been a member of The Medical Society of Virginia for twenty-three years.

Dr. Lee is survived by four daughters and one son, Dr. Claude Marshall Lee, Jr., of Hingham, Massachusetts.

Dr. Payne.

Dr. Robert Lee Payne, distinguished physician and surgeon, died at his home May 7, 1967. He was born December 4, 1882, in Lexington, N. C.

Moving to Norfolk with his parents in 1894, he attended the Norfolk Public Schools, the University of North Carolina from 1900 to 1901, and the University of Pennsylvania Medical School from 1901 to 1905, at which time he was awarded his M.D. degree. After interning a year at St. Agnes Hospital in Philadelphia, Dr. Payne returned to Norfolk and began practice of medicine in partnership with his father. This began a long and honorable career of 57 years, until he retired in 1963.

Possessed of a superb mind and marked talent as a surgeon, Dr. Payne was recognized at home and nationally as a master surgeon with exceptional initiative and resourcefulness, great courage and integrity. As a result of this he had many honors conferred upon him and took a very active part in the organization and functioning of medical and surgical societies and related auxiliaries. He served as Chief of the Surgical Staffs at Norfolk General Hospital, St. Vincent's and De Paul Hospitals, and for a long period of time was Chief Surgeon for the Norfolk-Southern Railway. He was a Past-President of the Norfolk County Medical Society, the Seaboard Medical Society and the Southern Surgical Association. He was for many years the Secretary of the Southern Surgical Association and had the responsibility of editing the publication of the transactions of that Society.

He was a Fellow of the American Surgical Association, a Fellow and member of the American College of Surgeons, one of the founders and a charter member of the American Board of Surgery. He was a member of The Medical Society of Virginia, American Medical Association, Southern Medical Association, Virginia Surgical Society, honorary member of the Medical Society of North Carolina, former Chairman of the Medical Advisory Board, Selective Service System for Norfolk during World War II.

Dr. Payne was a member and former Elder of the First Presbyterian Church, a member of the Norfolk Yacht & Country Club, Mastigouche Fish and Game Club of Quebec, Canada, and the Norfolk German Club. He was also one of the founding members of the Princess Anne Country Club.

Besides his wife, he is survived by five children. His other survivors are two sisters, thirteen grandchildren and three great grandchildren.

WHEREAS, the passing of our friend and colleague is recorded with deep regret let it be

RESOLVED, that a copy of this tribute be recorded in the minutes of the Society and a copy be transmitted to the family of the deceased. Be it therefore

RESOLVED, that a copy be transmitted to the Virginia Medical Monthly.

R. BRYAN GRINNAN, JR., M.D.
ROBERT E. MCALPINE, M.D.
C. C. SMITH, JR., M.D., *Chairman*

Dr. Lowenberg.

Dr. Eugene L. Lowenberg died June 8, 1967 after a brief illness. With his death Tidewater Virginia lost a dedicated physician and a distinguished surgeon.

winter 1968

DORSEY Season

A journal within a journal published quarterly in the interests of better medicine by Dorsey Laboratories, a division of The Wander Company, Lincoln, Nebraska 68501. Address communications to Keith W. Sehnert, M.D., Medical Director.





this issue: the cold in pregnancy...

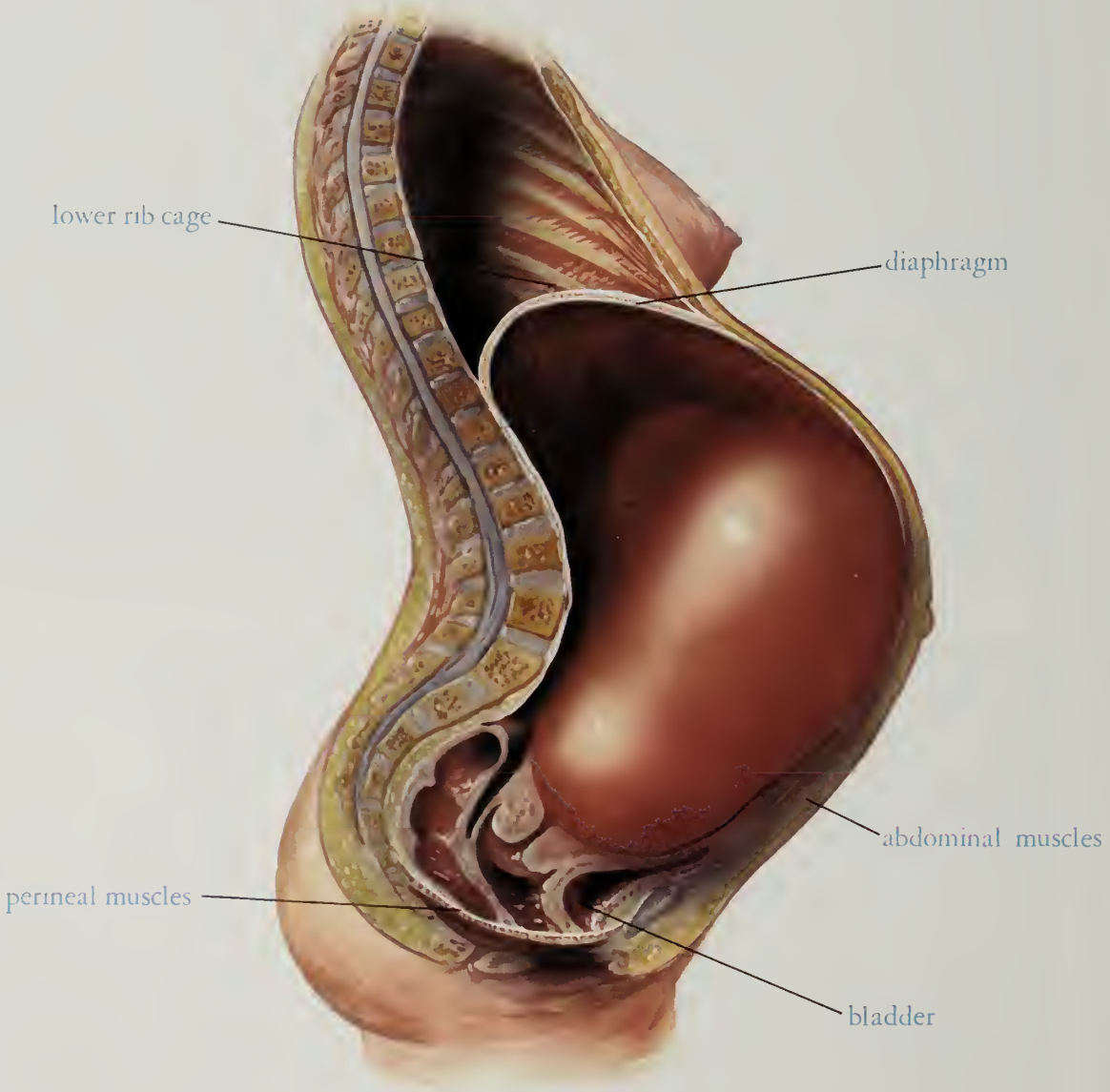
the cold in pregnancy

Frederick W. Goodrich, Jr., M.D.
Senior Obstetrician and Gynecologist, Lawrence and Memorial Hospital, New London, Connecticut

Since pregnancy and the common cold are both ubiquitous, it is not surprising that they often occur at the same time in the same patient. Incidence figures are hard to come by, but the chances are very good that any given pregnant woman will have a cold at some time during the nine months of her gestation. When she does, she will tell you that it is the worst cold she ever had. It seems to last longer and the symptoms are more distressing than they are in the non-pregnant.

For purposes of this discussion, the common cold is considered to be the well-known symptom complex which includes sore throat, stuffy nose, and a cough. Febrile states or extension of the disease process into the lower respiratory tree are not part of the common cold and will not be included in this discussion.

The clinical picture of a cold in pregnancy can be confused by a long-known physiological phenome-



non. Kiesselbach's area in the nose (on the anterior part of the nasal septum above the intermaxillary bone) becomes engorged, apparently due to hyperemia induced by the increased estrogen level which accompanies pregnancy. The amount of congestion can vary in degree from woman to woman. Some have very little congestion, others will have occasional nosebleeds from this area, still others will have symptomatic congestion to the degree that they will complain of having a "chronic" or constant cold.

ess well recognized is the occurrence of this type of hyperemia in any part of the nasopharyngeal mucosa, again in varying degree. Such swelling often produces a postnasal drip which, the patient will state, is present only when she is pregnant. Patients who do not have symptomatic congestion ordinarily, will find that when they do get a cold, he symptoms last much longer than those of a cold usually do. Occasionally, this hyperemia is responsible for closure of the medial end of the Eustachian ube; such patients will complain of "plugging" of he ears. Inspection of the ear drum will show a lepression which confirms the presence of Eustachian closure rather than wax in the canal which is he patient's diagnosis. Symptoms related to this hysiological congestion are more apt to occur in eavier smokers or those who have a history of llergic rhinitis, just as are the symptoms of the ommon cold. And when the cold does occur in regnancy, the symptoms are worse because of the nderlying congestion.

The pregnant woman with a cold is miserable for ther reasons, dependent somewhat on her parity nd the length of her gestation. As parity increases, o also does the relaxation of the abdominal and erineal musculature. The uterus, lying against a lack abdominal wall, and bearing down on relaxed erineal muscles, acts like a piston when the patient oughs, sneezes, or even blows her nose, pushing lown on the bladder. Stress incontinence during olds is almost the rule.

s the length of gestation increases, so does the size f the uterus. As it grows, it pushes the abdominal ontents above it and elevates the diaphragm. This esults eventually in a lateral displacement of the ower rib cage, often to a point at which the patient vill complain of soreness in this area. If such a

(Concluded on following page)

From a continuing study on nasal congestion...



timed to work while your patient does

A study begun in 1966 by the Department of Otolaryngology, Greater Baltimore Medical Center, has accumulated evidence that documents the effectiveness of Triaminic's timed-release action in the treatment of nasal congestion.

With its balanced formulation of an oral nasal decongestant and two antihistamines, Triaminic effected partial or complete relief in more than 82% of the 85 subjects treated. Clearing nasal obstruction. Reducing turbinate swelling. Making breathing easier.

It's a comforting thing to know that Triaminic really works.

Triaminic® *timed-release tablets*

Each timed-release tablet contains:

Phenylpropanolamine hydrochloride	50mg.
Pyrilamine maleate	25mg.
Pheniramine maleate	25mg.

Side effects: Occasional drowsiness, blurred vision, cardiac palpitations, flushing, dizziness, nervousness or gastrointestinal upsets. Precautions: The patient should be advised not to drive a car or operate dangerous machinery if drowsiness occurs. Use with caution in patients with hypertension, heart disease, diabetes or thyrotoxicosis.

(Advertisement)

patient has a cold, the coughing and sneezing will exaggerate this soreness. The diaphragmatic restriction caused by the enlarging uterus also makes breathing more difficult, particularly when the patient lies down. A cold compounds this difficulty.

Treating a cold in pregnancy requires two additional considerations. The patient should be told that the symptoms may last longer lest she become discouraged, and she should be reassured that the treatment will not damage the fetus. Although there are still some women who seem blissfully ignorant regarding matters of health (and these need to be cautioned not to take medication recommended by friends), most of our maternity patients are aware of the thalidomide tragedy. These women need constant reassurance that the symptoms of a cold can be treated, and that the medication is safe. Fortunately, many of the drugs which are of benefit have been in use long enough and widely enough so that such a statement can be offered. Occasionally one encounters a patient who is so anxious to avoid medication that she is extremely reluctant to take anything at all. Since cold is self-limited, it seems reasonable enough to give such a patient the choice and not to force drugs on her if she is obviously afraid of taking them.

The most distressing symptom of the common cold is the nasal congestion. This can be mitigated by the use of a decongestant which will help reduce the postnasal drip and the coughing caused by it. The kind of a cough which is described as a "tickling" in the throat and which is apt to occur when the patient goes to bed is usually due to nasopharyngeal congestion rather than inflammation in the lower respiratory tract.

There are many cough mixtures available. Most contain an antihistamine, a decongestant, and a cough "suppressant." Should cough suppression be desirable, it is important to remember not to use both a decongestant tablet and such a cough mixture as the patient would be getting a double dose of the decongestant. With some patients, the placebo effect of a cough syrup may be important and a liquid preparation might be substituted for an oral tablet.

For a sore throat, candy drops or lozenges at frequent intervals are soothing. It is not only unnecessary but unwise to use antibiotic lozenges or drops. The use of antibiotics in an uncomplicated cold is contraindicated and should be scrupulously avoided.

In summary, a cold in pregnancy is more severe and longer lasting. The treatment of the symptoms with local and systemic decongestants will make the patient more comfortable.

Apparently the cold is so common in pregnancy that it has received very little attention in the literature. References are almost non-existent and the few which are available add little to the common knowledge, are out-dated, or are not helpful. Thus the usual bibliography is not appended.



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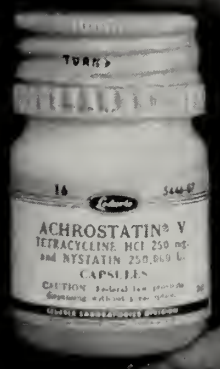
Each teaspoonful (5 ml.) contains:

Phenylpropanolamine hydrochloride	12.5 m
Pheniramine maleate	6.25 m
Pyrilamine maleate	6.25 m

For nasal congestion you can bring quick, lasting co fort to your little patients with Triaminic Syrup. You m occasionally encounter these side effects: drowsine blurred vision, cardiac palpitations, flushing, dizzine nervousness or gastrointestinal upsets. Precautions: t possibility of drowsiness should be considered by p tients engaged in mechanical operations requiring ale ness. Use with caution in patients with hypertensic heart disease, diabetes, or thyrotoxicosis.

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Tuinal helps wakeful patients fall asleep fast, stay asleep all night.

Indications: Tuinal is indicated for prompt and moderately long-acting hypnosis. It is not suitable for continuous daytime sedation.

Contraindications: Barbiturates should not be administered to anyone with a history of porphyria, nor should they be given in the presence of uncontrolled pain, because excitement may result.

Warning: May be habit-forming.

Precautions: Tuinal should be used cautiously in patients with decreased liver function, since prolongation of effect may occur.

Adverse Reactions: Idiosyncrasy, such as excitement, hangover, or pain, may appear. Hypersensitivity reactions occur in some patients, especially in those with asthma, urticaria, or angioneurotic edema.

Overdosage: C.N.S. depression. Symptoms—Depression of respiration and of superficial and deep reflexes, slight constriction of the pupils (in severe poisoning, dilation), decreased urine formation, lowered body temperature, coma. Treatment—Symptomatic and supportive (gastric lavage; intravenous fluids; maintenance of blood pressure, body temperature, and adequate respiration). Dialysis may speed removal of barbiturates from body fluids.



Dosage: 50-200 mg. (¾-3 grains) at bedtime.

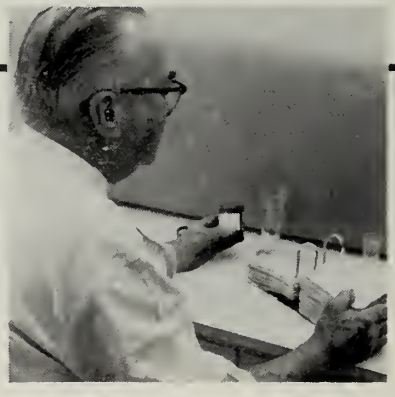
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Dr. Lowenberg was born in Norfolk, July 11, 1898. Following a boyhood accident which necessitated repeated operations on his right leg, he determined to become a surgeon. After graduating from the University of Virginia in 1920 and from the University of Virginia Medical School in 1923 and interning in New York City, he returned to Norfolk and devoted his life to service in his native community.

An early interest in the treatment of varicose veins led him into the burgeoning field of peripheral arterial surgery. He was a prodigious worker, and partly because of his love for his work and partly because of his extraordinary vitality, he was able to carry out long surgical procedures and to see numerous patients at his office as well. While he took pride and pleasure in his technical accomplishment, he never lost his concern for each patient as an individual and was available to every patient at any time.

He regularly read and studied numerous medical journals; he traveled widely in this country and abroad attending clinics, conferences, and meetings concerned with vascular surgery. He maintained his enthusiasm for advances in general and vascular surgery up to the time of his death. He was also an enthusiastic teacher and played an important part in the teaching programs at the local hospitals. He wrote and published in local, state, national, and international medical journals articles especially valuable because they reflected his personal experience with large numbers of patients.

Dr. Lowenberg had practiced locally since 1927. He was a past president of the Medical Staff of Norfolk General Hospital, Chief of the Vascular Surgical Service at both Norfolk General and De Paul Hospitals, and a member of the staffs of Leigh Memorial, Portsmouth General, Maryview, and Virginia Beach Hospitals. He was a lecturer and consultant to the Portsmouth Naval Hospital. He was a member of the Norfolk County Medical Society, The Medical Society of Virginia, Seaboard, Tri-State, Southern and American Medical Associations. He was a senior member of the International Cardiovascular Society, and honorary life member of the Pan American Medical Association, an honorary member of the Surgical Society of Madrid, Spain, and a Fellow of the American College of Surgeons and of the American College of Angiology. He was a member of the Virginia Surgical Association and the Southeastern Surgical Congress, and a Diplomate of the American Board of Surgery.

Dr. Lowenberg is survived by his widow and two sons.

Be It Resolved: That the Norfolk County Medical Society enter in its minutes these remembrances of Dr. Lowenberg and convey sympathy to his family.

Be It Further Resolved: That a copy be sent to the family and the Virginia Medical Monthly.

M. Richard Whitehill, M.D., *Chairman*
Arnold Zetlin, M.D.
Charles E. Davis, Jr., M.D.

Dr. Crosby.

Albert V. Crosby, M.D., died June 13, 1967, in the Emergency Room of De Paul Hospital of a myocardial infarction suffered earlier in the day while enroute to the hospital to make rounds.

Dr. Crosby was born in Norfolk on October 5, 1895. Pre-medical education was obtained at Mount Saint Joseph's College in Baltimore, Maryland. In 1913 he entered Hahnemann Medical College and graduated in 1917. Dr. Crosby interned at Hahnemann Hospital and later served a residency at Abingdon Memorial Hospital, Abingdon, Pennsylvania.

In 1917 he was commissioned First Lieutenant, Medical Reserve, and served a tour of duty at Camp Gordon, Georgia. He returned to civilian life February 3, 1919. Early in 1919 he received his license to practice medicine in Virginia and engaged in the private practice of General Surgery and Obstetrics and Gynecology in this area until his death.

Dr. Crosby held membership on the staffs of De Paul Hospital and Leigh Memorial Hospital. He had actively participated in the teaching programs at Saint Vincents, later De Paul Hospital, Kings Daughters Clinic for Children, and served as a visiting physician in the Tuberculosis Clinic under Charles Grandy, M.D.

His interests in community activities were catholic and he gave freely of his time and talents. He served the American Red Cross as Chairman of the First Aid and Lifesaving Committee, and later as Chairman of the Disaster Committee. In this latter capacity, he organized and supervised a disaster warning program during the great hurricane of 1938. By strategically placing many two-way radio transmitters throughout the Norfolk-Willoughby area, this operation was instrumental in lessening the loss of life and property damage during this disaster.

In World War II he assisted in the formation of and was active in recruiting members to serve in the Canteen Service for the Airplane Watch. He served as Co-Chairman of the Civilian Defense Medical Service, and at the time of his death, he held the post of Deputy Chief of Medical Service of Civil Defense.

From 1943 to 1948 Dr. Crosby served as President of the Travelers Aid Society. He was a past Vice-Commander of Post 67, American Legion. At one time he served as Commanding Officer of 110th Am-

bulance Company, 104th Medical Regiment, Virginia National Guard. This unit was organized by him at the request of the Adjutant General of Virginia. His interest in the Norfolk Fire Department was lifelong. For years he served as Surgeon, Norfolk Fire Division, and at the time of his death was Honorary Chief, Norfolk Fire Division.

Dr. Crosby's dedication to lifesaving was further manifested by his activities as Chairman of the Cardiac-Pulmonary Resuscitation Committee of the Virginia Heart Association. As its representative, he traveled throughout the State demonstrating cardiopulmonary resuscitative measures and trained many people in widely scattered areas to serve as local chairmen.

In 1937 Dr. Crosby was surgeon of a field hospital at the Boy Scouts of American Jamboree in Washington, D. C. He served as Chairman of the Health and Safety Committee of the Boy Scouts of America.

Together with Dr. Frank Redwood, Dr. Leslie H. Barber and Miss Sue Slaughter, he was a member of the committee instrumental in organizing the Norfolk Mental Health Clinic.

Saint Mary's Infant Home listed him on its Board of Directors, as did the Catholic Family and Child Service.

In 1965 a mobile medical unit equipped with oxygen, resuscitative equipment, operating table and surgical equipment for emergencies was donated, in Dr. Crosby's name, to the City of Norfolk. This dream came true due to the efforts and support of the Beta Sigma Phi Sorority and the Norfolk Fire Division.

In April 1966 Dr. Crosby was honored with the presentation of a civic award, given annually to a Catholic layman for contributions to the community, by the Knights of Columbus. He was a member of the Blessed Sacrament Catholic Church in Norfolk, Virginia, and a member of the Knights of Columbus. He is survived by a son, a daughter, four grandchildren and one great grandchild.

Dr. Crosby never refused a task which in his opinion had merit, even though he was already burdened with many voluntary community activities. His own physical disabilities were never a deterrent to accept additional tasks. His personal charities were many, though never publicized. He respected his colleagues, enjoyed the practice of medicine, and won the admiration and respect of his peers in the profession and his patients. His devotion to causes he thought were right can best be epitomized by repeating a chance remark by an unknown clinic patient overheard outside the emergency room door at the time of his death, who said, "There goes one of the last of a dying breed of physicians."

His presence will be sorely missed by all who knew him and his record of service to this community will not be soon forgotten or equalled.

THEREFORE, BE IT RESOLVED that a copy of these Resolutions be spread on the minutes of the Norfolk County Medical Society and copies be sent to his son, daughter, and the Virginia Medical Monthly.

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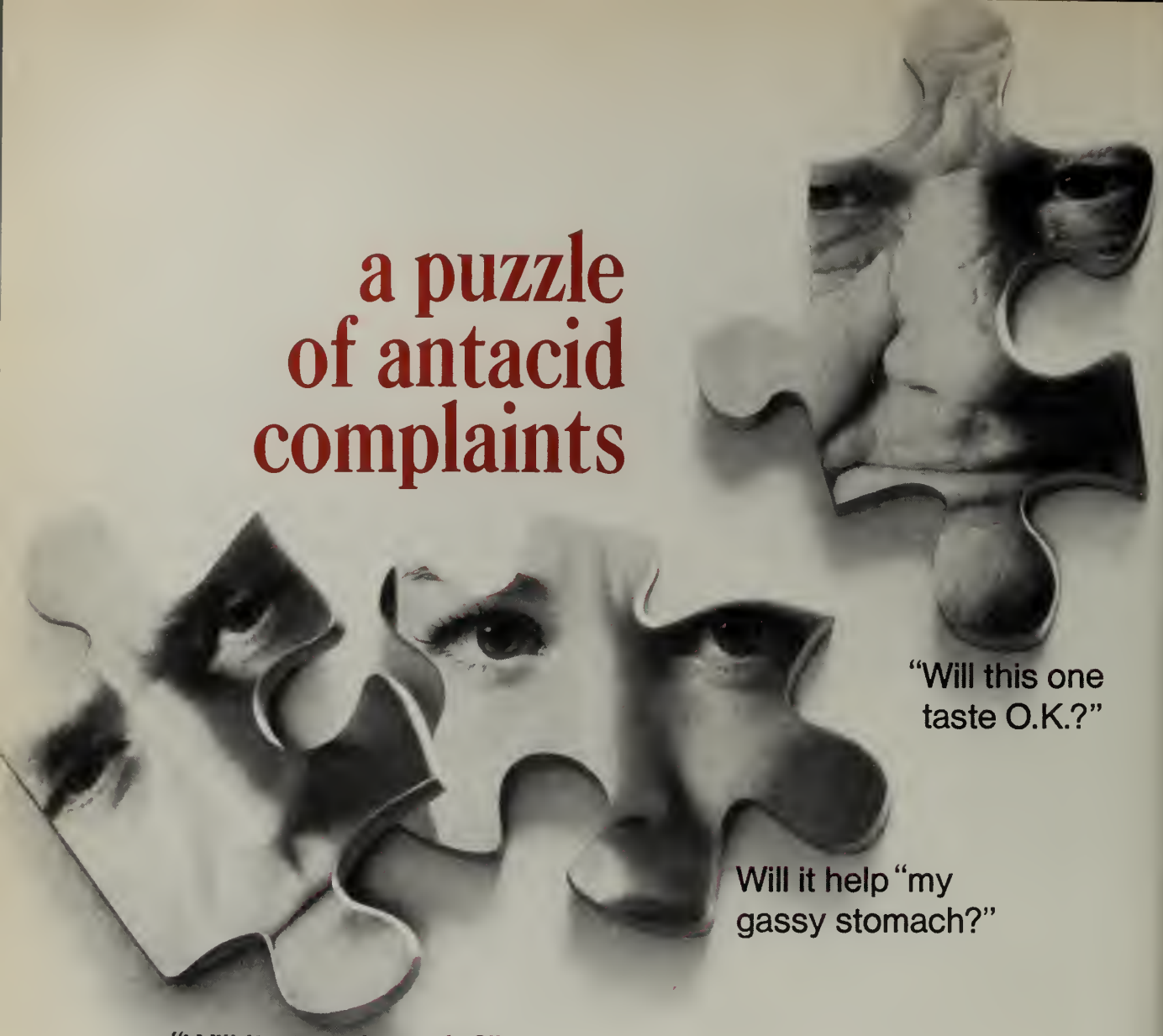
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References: 1. Hoon, J. R.: Arch. Surg. *93*:467 (Sept.) 1966.
2. Danhof, I. E., Personal communication.

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ERYTHROCIN®-SULFAS Granules (Erythromycin ethyl succinate-trisulfapyrimidines granules for oral suspension)

87 patients were treated[1,2]—all children, ages four months to 15 years.

The diagnoses were multiple in some cases and were chiefly bacterial infections of the respiratory tract.

Dosage was maintained from three to 10 days; average treatment was five days. All of the ill children accepted the orange-flavored suspension favorably.

53 were clinically cured within 72 hours, while 32 showed partial relief within the same time, and subsequent clinical cure.

701358

A clinical cure rate of 97.7%

1. Case Reports on File, Dept. Clin. Development, Abbott Laboratories.
2. Polley, R.F.L., Use of Erythromycin-Sulfas in Office Practice, Western Med., 7:177, July, 1966.

ABBOTT

Brief Summary on next page

ERYTHROCIN®-SULFAS
Brief Summary

Contraindications: Known sensitivity to erythromycin or sulfonamides. Because of the possibility of kernicterus with sulfonamides, do not use in pregnancy at term, premature or newborn infants.

Warnings: As with other forms of sulfonamide therapy, carefully evaluate patients with liver or kidney damage, urinary obstruction, or blood dyscrasia. Deaths have been reported from hypersensitivity reactions and blood dyscrasias following use of sulfonamides. Perform blood counts and liver and kidney function tests if used repeatedly at close intervals or for long periods.

Precautions, Side Effects: Occasionally mild abdominal discomfort, nausea or vomiting may occur with erythromycin, generally controlled by reduction of dosage. Mild allergic reactions (such as urticaria and other skin rashes) may occur. Serious allergic reactions have been extremely infrequent. Use sulfonamides with caution in patients with a history of allergy. Assure adequate fluid intake to prevent crystalluria and institute alkali therapy if indicated. If overgrowth of nonsusceptible organisms occurs, withdraw the drug and institute appropriate treatment. If a patient should show signs of hypersensitivity, appropriate countermeasures (e.g. epinephrine, steriods, etc.) should be administered and the drug withdrawn.

Adverse Reactions: Sulfonamide therapy may be associated with headache, nausea, vomiting, urticaria, diarrhea, hepatitis, pancreatitis, blood dyscrasias, neuropathy, drug fever, skin rash, injection of the conjunctiva and sclera, petechiae, purpura, hematuria and crystalluria.

Side effects due to erythromycin are infrequent, but occasional abdominal discomfort, nausea, or vomiting, urticaria and other skin rashes may occur.

Supplied: The Granules for Oral Suspension come in bottles of 60 ml. and 150 ml. The Chewable tablets are in bottles of 50. Each 5-ml. teaspoonful of reconstituted Granules or each Chewable tablet provides erythromycin ethyl succinate equivalent to 125 mg. of erythromycin activity and 167 mg. of each of sulfadiazine, sulfamerazine and sulfamethazine.

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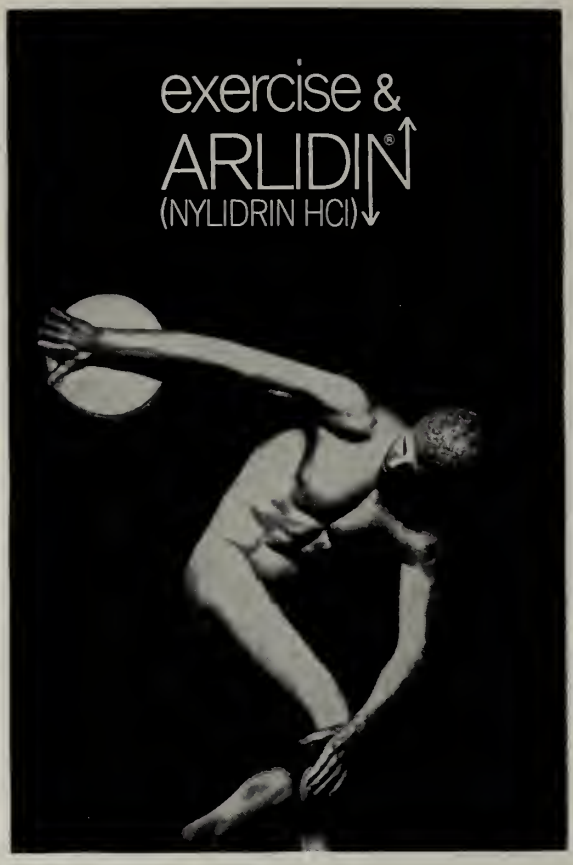


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Sources of sinus headache



REGION OF SENSATION

FRONTAL SINUS—headache localized over the frontal region, commonly beginning in the morning. Eyes painful to touch, may be slight swelling over eyes, puffy eyelids.
SPHENOID SINUS—most difficult to diagnose. Headache or sense of pressure over vertex. May be pain in ear or mastoid region.



MAXILLARY SINUS—headache and tenderness under eyes at top of cheekbones, usually starting in early afternoon. Possible swelling of cheek. Pain in jaw and back teeth.
ETHMOID SINUS—headache, tightness in upper part of bridge of nose and well down its sides. Pain, deep-seated behind the eyes and in inner corners of the eyes.



TURBINATES—pain along medial and lateral walls of the inside of the nose. Headache in frontal region, along supraorbital ridge, a feeling of fullness in the head.

Source of symptomatic relief

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Precautions: Instruct patients not to drive or operate machinery if drowsiness occurs. Use with caution in patients with thyroid disease, heart disease, hypertension, diabetes or kidney disease. Excessive dosage or prolonged use may cause kidney damage.

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One by one the family's downed Because the G.I. bug's around

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Whether it's a 24-hour "bug", a food problem, or simply nervousness and anxiety, Parepectolin will bring the diarrhea under control until etiology can be determined. In some cases, Parepectolin may be all the therapy necessary.

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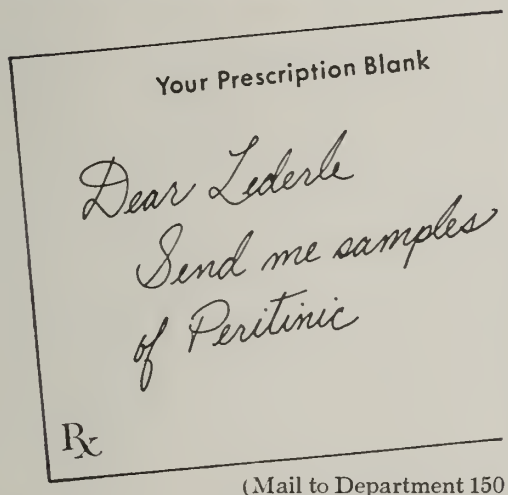
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Contains opium (¼ grain) 15 mg. per fluid ounce.
warning: may be habit forming
Pectin ..... (2½ grains) 162 mg.
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* anticostive, *adj.* (*anti* opposed to + *costive* causing constipation.) Against constipation. (Now isn't that a good idea in an iron-containing hematinic?)

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Vitamin B_6	7.5 mg
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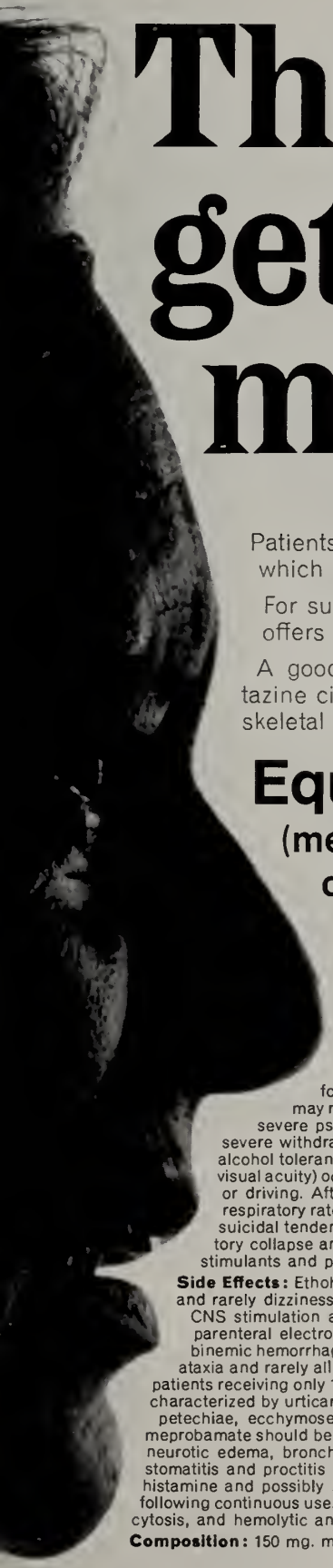
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This pain is getting on my nerves.

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A good choice is often EQUAGESIC® (meprobamate and ethoheptazine citrate with aspirin). It helps relieve pain. And anxiety. And skeletal muscle spasm as related to pain or anxiety and tension.

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(meprobamate and ethoheptazine citrate with aspirin)

Wyeth®

Contraindications: History of sensitivity or severe intolerance to aspirin or meprobamate.

Warnings: USE IN PREGNANCY: Safety for use during pregnancy or lactation has not been established; therefore it should be used in pregnant patients or women of child-bearing age only when the physician judges its use essential to the patient's welfare.

Precautions: Keep out of reach of children. Not recommended for patients 12 years old or less. Carefully supervise dose and amounts prescribed, especially for patients prone to overdose themselves. Excessive prolonged use of meprobamate may result in dependence or habituation in susceptible persons—as alcoholics, ex-addicts, severe psychoneurotics. Withdraw gradually after prolonged high dosage to avoid possibly severe withdrawal reactions including epileptiform seizures. Warn patients of possible reduced alcohol tolerance. If drowsiness, ataxia or visual disturbances (impairment of accommodation and visual acuity) occur, reduce dose. If symptoms persist, caution patients against operating machinery or driving. After meprobamate overdose, prompt sleep, reduction of blood pressure, pulse and respiratory rates to basal levels, and hyperventilation are reported. Give cautiously to patients with suicidal tendencies. Treat attempted suicide (has resulted in coma, shock, vasomotor and respiratory collapse and anuria) with immediate gastric lavage and appropriate supportive therapy (CNS stimulants and pressor amines as indicated).

Side Effects: Ethoheptazine and aspirin may occasionally cause nausea, vomiting, epigastric distress, and rarely dizziness. Overdosage may result in CNS depression (drowsiness and lightheadedness) or CNS stimulation and salicylate intoxication (requires induced vomiting or gastric lavage, specific parenteral electrolyte therapy for ketoacidosis and dehydration, and observation for hypoprothrombinemic hemorrhage [usually requires whole blood transfusions]). Meprobamate may cause drowsiness, ataxia and rarely allergic or idiosyncratic reactions. These reactions, sometimes severe, can develop in patients receiving only 1 to 4 doses who have had no previous contact with meprobamate. Mild reactions are characterized by urticarial or erythematous maculopapular rash. Acute nonthrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever have been reported. If allergic reaction occurs, meprobamate should be stopped and not reinstituted. Severe reactions, observed very rarely, include angioneurotic edema, bronchial spasms, fever, fainting spells, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case) and hyperthermia. Treat symptomatically such as with epinephrine, antihistamine and possibly hydrocortisone. A few cases of leucopenia, usually transient, have been reported following continuous use. Rarely, cases of aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis, and hemolytic anemia have been reported; almost always, in the presence of known toxic agents.

Composition: 150 mg. meprobamate, 75 mg. ethoheptazine citrate and 250 mg. aspirin per tablet.

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in moderate hypertension and
poorly controlled mild hypertension
(when diuretics or sedatives are inadequate, for example)

When your patients expect a lot...

like relief from hypertensive symptoms
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chlorthalidone 50 mg., reserpine 0.25 mg.

Indications: Hypertension. **Contraindications:** History of mental depression, hypersensitivity, and most cases of severe renal or hepatic diseases. **Warning:** With the administration of enteric-coated potassium supplements, which should be used only when adequate dietary supplementation is not practical, the possibility of small-bowel lesions (obstruction, hemorrhage, and perforation) should be kept in mind. Surgery for these lesions has frequently been required and deaths have occurred. Discontinue coated potassium-containing formulations immediately if abdominal pain, distention, nausea, vomiting, or gastrointestinal bleeding occur. Discontinue one week before electroshock therapy, and if depression or peptic ulcer occurs. *Use in pregnancy:* Regroton should be used in pregnant patients or in women of childbearing potential only when, in the judgment of a physician, its use is deemed essential to the welfare of the patients; adverse reactions (thrombocytopenia, hyperbilirubinemia, altered carbohydrate metabolism, etc.) are potential problems in the newborn.
Precautions: Antihypertensive therapy with Regroton should always be initiated cautiously in postsympathectomy patients and in patients receiving ganglionic blocking agents, other potent antihypertensive drugs, or curare. Reduce dosage of concomitant antihypertensive agents by at least one-half. To avoid hypotension during surgery, discontinue Regroton therapy two weeks prior to elective surgical procedures. In emergency surgery, use, if needed, anticholinergic or adrenergic drugs or other supportive measures as indicated. Because of the possibility of progression of renal damage, periodic kidney function tests are indicated. Discontinue if the BUN rises or liver dysfunction is aggravated. Hepatic coma may be precipitated. Electrolyte imbalance, sodium and/or potassium depletion may occur. If potassium depletion should occur during therapy, Regroton should be discontinued and potassium supplements given, provided the patient does not have marked oliguria. Take particular care in cirrhosis or severe ischemic heart disease and in patients receiving corticosteroids, ACTH, or digitalis. Salt restriction is not recommended. Use cautiously in patients with ulcerative colitis or gallstones (biliary colic may be precipitated). Bronchial asthma may occur in susceptible patients. **Adverse Reactions:** The drug is generally well tolerated. The most frequent side effects are nausea, gastric irritation, vomiting, diarrhea, constipation, muscle cramps, headache, dizziness and acute gout. Other potential side effects include angina pectoris, anxiety, depression, bradycardia and ectopic cardiac rhythms (especially when used with digitalis), drowsiness, dull sensorium, hyperglycemia and glycosuria, hyperuricemia, lassitude, restlessness, transient myopia, impotence or dysuria, orthostatic hypotension which may be potentiated when chlorthalidone is combined with alcohol, barbiturates or narcotics, leukopenia, aplastic anemia, skin rashes, thrombocytopenia, agranulocytosis, nasal stuffiness, increased gastric secretions, nightmare, purpura, urticaria, ecchymosis, weakness, uveitis, optic atrophy and glaucoma, and pruritus. Eruptions and/or flushing of the skin, a reversible paralysis agitans-like syndrome, blurred vision, conjunctival injection, increased susceptibility to colds, dyspnea, weight gain, decreased libido, dryness of the mouth, deafness, anorexia, and pancreatitis when epigastric pain or unexplained G.I. symptoms develop after prolonged administration. Jaundice, xanthopsia, paresthesia, photosensitization and necrotizing angiitis are possible. **Average Dosage:** One tablet daily with breakfast. **Availability:** Pink, single-scored tablets in bottles of 100 and 1000. (B) 46-600-B
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When the talk turns to oral contraceptives, it makes medical sense to remember low-dose Norinyl®-1.

(norethindrone 1mg. ē mestranol 0.05mg.)

Turn page for contraindications, precautions and side effects.



Reduction of oral contraceptive dosage to the lowest effective levels is a well-accepted principle of conservative medical practice. In keeping with this view, Norinyl is now also available as Norinyl-1, containing exactly one half the previous dosage of norethindrone and mestranol. Clinical experience has established that effective fertility control can be achieved with the same degree of reliability and safety with new Norinyl-1 when taken as directed.

What about switching patients from higher dosage forms?

In transferring patients to low-dose Norinyl-1 from higher-dosage oral contraceptives, some breakthrough bleeding may occur in the early cycles. In the majority of cases the bleeding episode is mild and self-limited. The long-term advantages of the lower dosage form should be weighed against the inconvenience of possible breakthrough bleeding in the individual patient.

Contraindications: 1. Patients with thrombophlebitis or with a history of thrombophlebitis or pulmonary embolism. 2. Liver dysfunction or disease. 3. Patients with known or suspected carcinoma of the breast or genital organs. 4. Undiagnosed vaginal bleeding.

Warnings: 1. Discontinue medication pending examination if there is sudden partial or complete loss of vision or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions, medication should be withdrawn. 2. Since the safety of NORINYL-1 (norethindrone 1 mg. with mestranol 0.05 mg.) in pregnancy has not been demonstrated, it is recommended that for any patient who has missed two consecutive periods, pregnancy should be ruled out before continuing the contraceptive regimen. If the patient has not adhered to the prescribed schedule, the possibility of pregnancy should be considered at the time of the first missed period. 3. Detectable amounts of the active ingredients in oral contraceptives have been identified in the milk of mothers receiving these drugs. The significance of this dose to the infant has not been determined.

Precautions: 1. The pretreatment physical examination should include special reference to breast and pelvic organs, as well as a Papanicolaou smear. 2. Endocrine and possibly liver function tests may be affected by treatment with NORINYL-1. Therefore, if such tests are abnormal in a patient taking NORINYL-1 it is recommended that they be repeated after the drug has been withdrawn for two months. 3. Under the influence of estrogen-progestogen preparations, preexisting uterine fibromyomata may increase in size. 4. Because these agents may cause some degree of fluid retention, conditions that may be influenced by this factor, such as epilepsy, migraine, asthma, cardiac or renal dysfunction, require careful observation. 5. NORINYL-1 should be used with caution in patients with a history of cerebrovascular accident. 6. In relation to breakthrough bleeding, and as in all cases of irregular bleeding *per vaginam*, nonfunctional causes should be borne in mind. In cases of undiagnosed vaginal bleeding, adequate diagnostic measures are indicated. 7. Patients with a history of psychic depression should be carefully observed and the drug discontinued if the depression recurs to a serious degree. 8. Any possible influence of prolonged NORINYL-1 therapy on pituitary, ovarian, adrenal, hepatic or uterine function awaits further study. 9. A decrease in glucose tolerance has been observed in a small percentage of patients on oral contraceptives. The mechanism of this decrease is obscure. For this reason, diabetic patients should be carefully observed while receiving NORINYL-1 therapy. 10. Because of the occasional occurrence of thrombophlebitis and pulmonary embolism in patients taking oral contraceptives, the physician should be alert to the earliest manifestations of the disease. 11. Because of the effects of estrogens on epiphyseal closure, NORINYL-1 should be used judiciously in young patients in whom bone growth is not complete. 12. The age of the patient constitutes no absolute limiting factor, although treatment with NORINYL-1 may mask the onset of the climacteric. 13. The pathologist should be advised of NORINYL-1 therapy when relevant specimens are submitted.

Side effects observed in patients receiving oral contraceptives: The following adverse reactions have been observed in patients receiving oral contraceptives: nausea, vomiting, gastrointestinal symptoms (such as abdominal cramps and bloating), breakthrough bleeding, spotting, change in menstrual flow, amenorrhea, edema, chloasma or melasma, breast changes (tenderness, enlargement and secretion), change in weight (increase or decrease), changes in cervical erosion and cervical secretion, suppression of lactation when given immediately postpartum, cholestatic jaundice, migraine, rash (allergic), rise in blood pressure in susceptible individuals, mental depression.

Although the following side effects have been reported in users of oral contraceptives, no cause and effect relationship has been established: anovulation post-treatment, premenstrual-like syndrome, change in libido, changes in appetite, cystitis-like syndrome, headache, nervousness, dizziness, fatigue, backache, hirsutism, loss of scalp hair, erythema multiforme, erythema nodosum, hemorrhagic eruption, itching.

The following occurrences have been observed in users of oral contraceptives (a cause and effect relationship has been neither established nor disproved): thrombophlebitis, pulmonary embolism, neuro-ocular lesions.

The following laboratory results may be altered by the use of oral contraceptives: increased sulfobromophthalein and other hepatic function tests, coagulation tests (increase in prothrombin, Factors VII, VIII, IX and X), thyroid function (increase in PBI and butanol extractable protein-bound iodine and decrease in T^3 values), metyrapone test, pregnanediol determination.

norethindrone — an original steroid from
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LABORATORIES INC., PALO ALTO, CALIF.

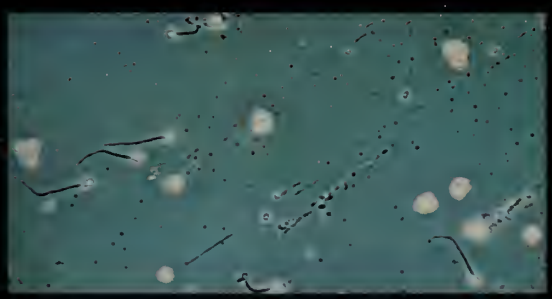
Here's why Norinyl-1 makes medical sense.

The effectiveness of Norinyl-1 as a low-dose oral contraceptive may be explained by its possible multiple action. In addition to its primary action of suppression of ovulation, Norinyl-1 may offer additional protective mechanisms... (1) creation of a cervical mucus that may be hostile to sperm penetration, and (2) development of an endometrium that may be out of phase with nidation. These effects are illustrated below.

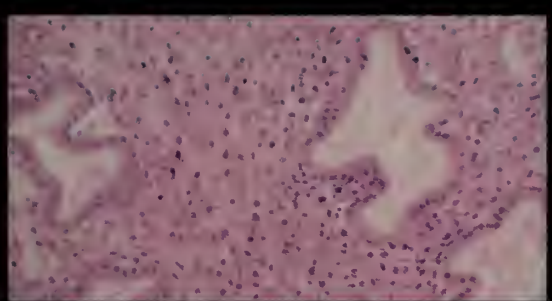
Untreated Patient



Cervical mucus at midcycle is usually thin and watery, with Spinnbarkeit (stretchability) of 15 to 20 cm.



Spermatozoa appear healthy, active, freemoving.

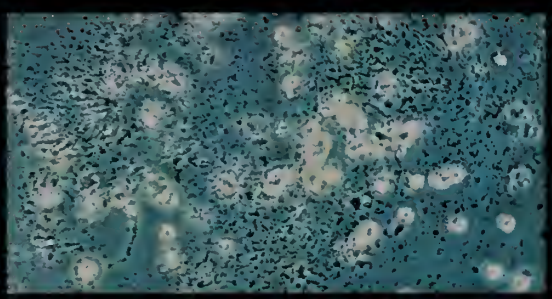


Endometrium of untreated patient is receptive to the fertilized ovum during secretory phase.

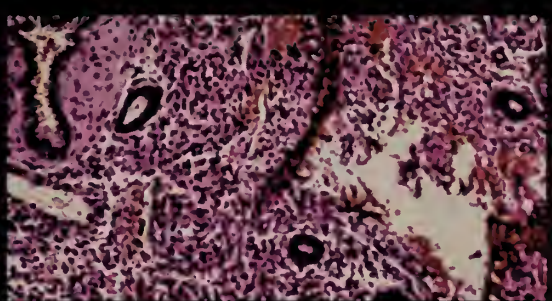
Norinyl-1 Patient



Cervical mucus at midcycle is scanty, viscous—with Spinnbarkeit of 1 cm. or less.



Immobile spermatozoa as they appear in cervical mucus taken from patient treated with Norinyl-1.



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fluocinolone acetonide





Turn page for contraindications, precautions and side effects

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Product Information

Contraindications: Tuberculous, fungal, and most viral lesions of the skin (including herpes simplex, vaccinia, and varicella). Not for ophthalmic use. Contraindicated in individuals with a history of hypersensitivity to any of the components. *Precautions:* Synalar preparations are virtually nonsensitizing and nonirritating. However, the solution may produce burning or stinging when applied to denuded or fissured areas. In some patients with dry lesions, the solution may increase dryness, scaling or itching. Where severe local infection or systemic infection exists, the use of systemic antibiotics should be considered, based on susceptibility testing. While topical steroids have not been reported to have an adverse effect on pregnancy, the safety of their use on pregnant females has not absolutely been established. Therefore, they should not be used extensively on pregnant patients, in large amounts, or for prolonged periods of time. *Side Effects:* Side effects are uncommon with topical corticosteroids. As with all drugs, however, a few patients may react unfavorably to Synalar under certain conditions. In such cases the agent should be discontinued and appropriate measures taken. *Availability:* Synalar (fluocinolone acetonide) Cream 0.025%—5, 15 and 60 Gm. tubes and 425 Gm. jars. Cream 0.01%—15, 45 and 60 Gm. tubes and 120 Gm. jars. Solution 0.01%—20 and 60 cc. plastic squeeze bottles. Ointment 0.025%—15 and 60 Gm. tubes. Neo-Synalar® (neomycin sulfate 0.5% [0.35% neomycin base], fluocinolone acetonide 0.025%) Cream—5, 15 and 60 Gm. tubes.

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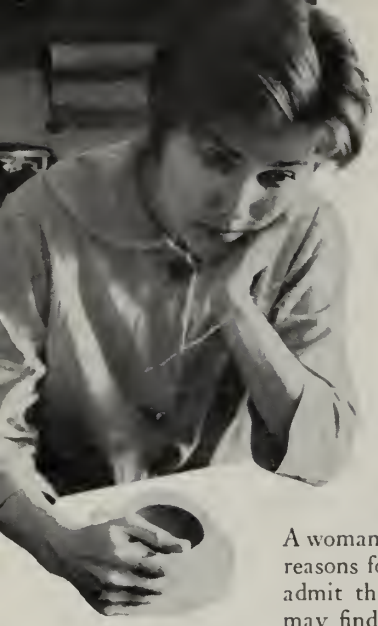
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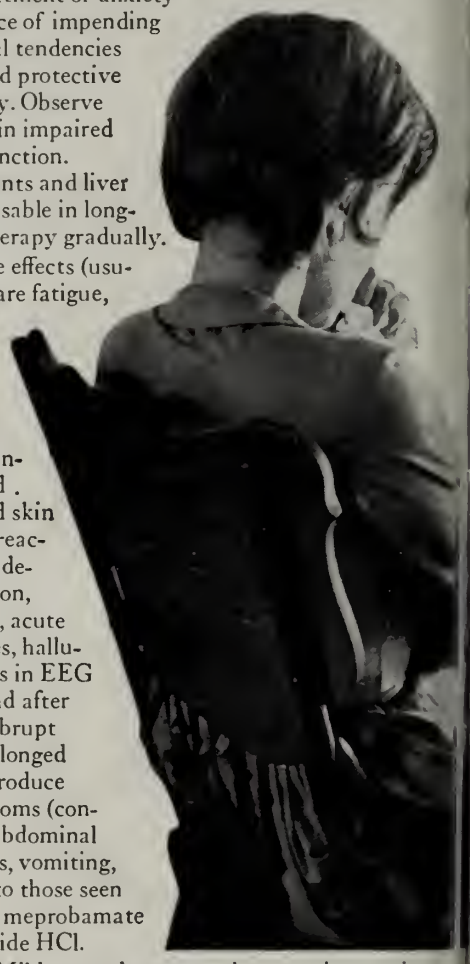


Before prescribing, please consult complete product information, a summary of which follows:

Contraindications: Infants, patients with history of convulsive disorders, glaucoma or known hypersensitivity to drug.

Warning: Not of value in the treatment of psychotic patients, and should not be employed in lieu of appropriate treatment.

Precautions: Limit dosage to smallest effective amount in elderly or debilitated patients (not more than 1 mg, one or two times daily initially) to preclude ataxia or oversedation, increasing gradually as needed or tolerated. As is true of all CNS-acting drugs, until correct maintenance dosage is established, advise patients against possibly hazardous procedures requiring complete mental alertness or physical coordination. Driving during therapy not recommended. In general, concurrent use with other psychotropic agents is not recommended. If such combination therapy is used, carefully consider individual pharmacologic effects–particularly with known compounds which may potentiate action of Valium (diazepam), such as phenothiazines, barbiturates, MAO inhibitors and other antidepressants. Advise patients against simultaneous ingestion of alcohol or other CNS depressants. Safe use in pregnancy not established. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Observe usual precautions in impaired renal or hepatic function. Periodic blood counts and liver function tests advisable in long-term use. Cease therapy gradually.



Side Effects: Side effects (usually dose-related) are fatigue, drowsiness and ataxia. Also reported: mild nausea, dizziness, blurred vision, diplopia, headache, incontinence, slurred speech, tremor and skin rash; paradoxical reactions (excitement, depression, stimulation, sleep disturbances, acute hyperexcited states, hallucinations); changes in EEG patterns during and after drug treatment. Abrupt cessation after prolonged overdosage may produce withdrawal symptoms (convulsions, tremor, abdominal and muscle cramps, vomiting, sweating) similar to those seen with barbiturates, meprobamate and chlordiazepoxide HCl.

Dosage: *Adults:* Mild to moderate psychoneurotic reactions, to 5 mg b.i.d. or t.i.d.; severe psychoneurotic reactions, 5 to 1 mg t.i.d. or q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 2 hours, then 5 mg t.i.d. or q.i.d. as needed; muscle spasm wit cerebral palsy or athetosis, 2 to 10 mg t.i.d. or q.i.d. *Geriatr patients:* 1 or 2 mg/day initially, increase gradually as neede and tolerated. (See Precautions.)

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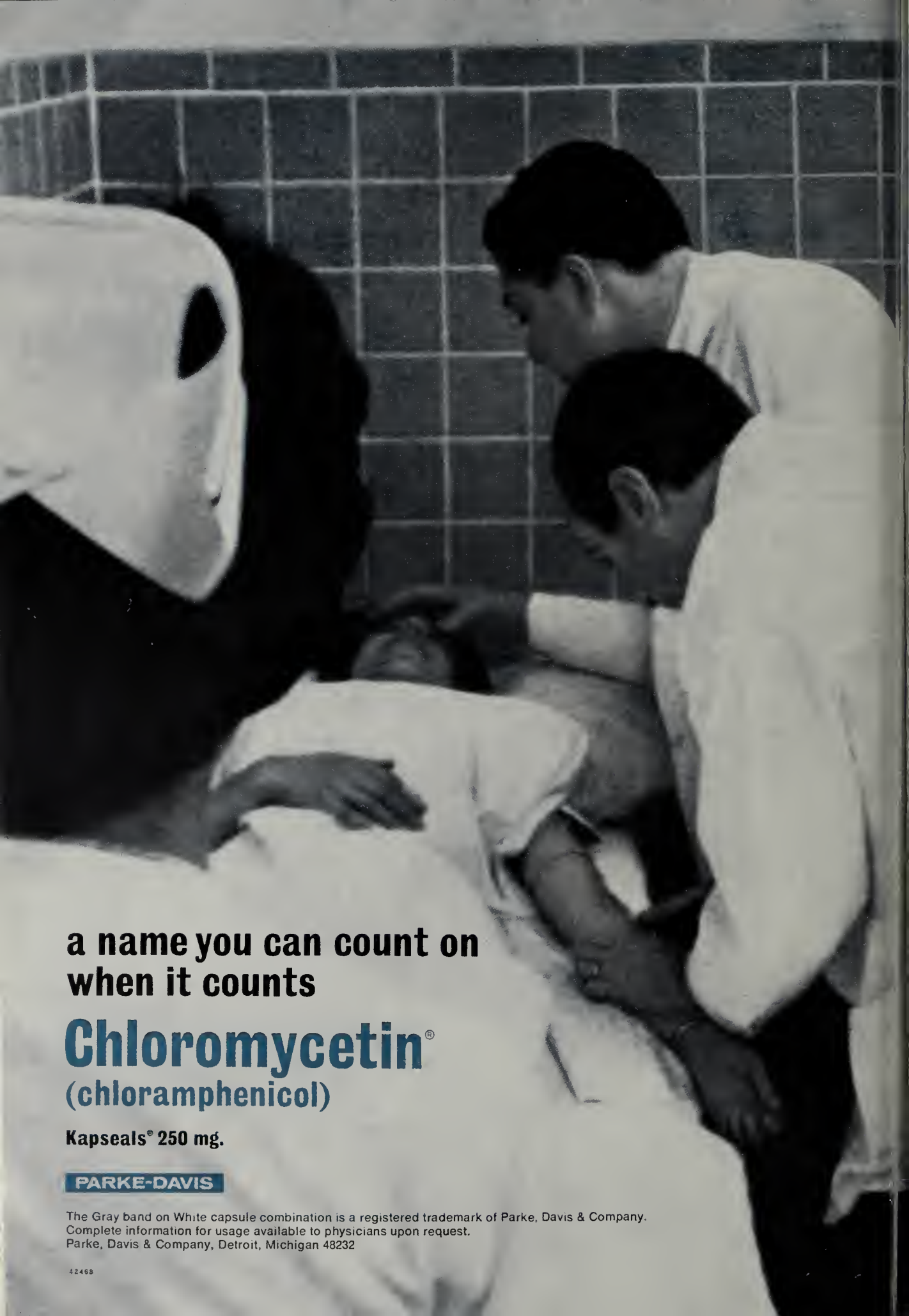
MEDICAL MONTHLY

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Precautions—Overgrowth of nonsusceptible organisms may occure. Constant observation is essential. If new infections appear, appropriate measures should be taken. In infants, increased intracranial pressure with bulging fontanels has been observed. All signs and symptoms have disappeared rapidly upon cessation of treatment.

Side Effects—Gastrointestinal system—anorexia, nausea, vomiting, diarrhea, stomatitis, glossitis, enterocolitis, pruritus ani. Skin—maculopapular and erythematous rashes. A rare case of exfoliative dermatitis has been reported. Photosensitivity; onycholysis and discoloration of the nails (rare). Kidney—rise in BUN, apparently dose related. Transient increase in urinary output, sometimes accompanied by thirst (rare). Hypersensitivity reactions—urticaria, angioneurotic edema, anaphylaxis. Teeth— dental staining (yellow-brown) in children of mothers given this drug during the latter half of pregnancy, and in children given the drug during the neonatal period, infancy and early childhood. Enamel hypoplasia has been seen in a few children. If adverse reaction or idiosyncrasy occurs discontinue medication and institute appropriate therapy.

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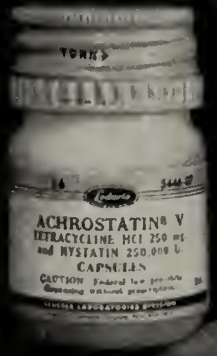
*As shown by *in vitro* studies.
1. Crunden, A. B., Jr., and Davis, W. A.: Am. J. Obst. & Gynec. *65*:311 (Feb.) 1953.



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(norethindrone 1mg. & mestranol 0.05mg.)

Turn page for contraindications, precautions and side effects.



Reduction of oral contraceptive dosage to the lowest effective levels is a well-accepted principle of conservative medical practice. In keeping with this view, Norinyl is now also available as Norinyl-1, containing exactly one half the previous dosage of norethindrone and mestranol. Clinical experience has established that effective fertility control can be achieved with the same degree of reliability and safety with new Norinyl-1 when taken as directed.

What about switching patients from higher dosage forms?

In transferring patients to low-dose Norinyl-1 from higher-dosage oral contraceptives, some breakthrough bleeding may occur in the early cycles. In the majority of cases the bleeding episode is mild and self-limited. The long-term advantages of the lower dosage form should be weighed against the inconvenience of possible breakthrough bleeding in the individual patient.

Contraindications: 1. Patients with thrombophlebitis or with a history of thrombophlebitis or pulmonary embolism. 2. Liver dysfunction or disease. 3. Patients with known or suspected carcinoma of the breast or genital organs. 4. Undiagnosed vaginal bleeding.

Warnings: 1. Discontinue medication pending examination if there is sudden partial or complete loss of vision or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions, medication should be withdrawn. 2. Since the safety of NORINYL-1 (norethindrone 1 mg. with mestranol 0.05 mg.) in pregnancy has not been demonstrated, it is recommended that for any patient who has missed two consecutive periods, pregnancy should be ruled out before continuing the contraceptive regimen. If the patient has not adhered to the prescribed schedule, the possibility of pregnancy should be considered at the time of the first missed period. 3. Detectable amounts of the active ingredients in oral contraceptives have been identified in the milk of mothers receiving these drugs. The significance of this dose to the infant has not been determined.

Precautions: 1. The pretreatment physical examination should include special reference to breast and pelvic organs, as well as a Papanicolaou smear. 2. Endocrine and possibly liver function tests may be affected by treatment with NORINYL-1. Therefore, if such tests are abnormal in a patient taking NORINYL-1 it is recommended that they be repeated after the drug has been withdrawn for two months. 3. Under the influence of estrogen-progestogen preparations, preexisting uterine fibromyomata may increase in size. 4. Because these agents may cause some degree of fluid retention, conditions that may be influenced by this factor, such as epilepsy, migraine, asthma, cardiac or renal dysfunction, require careful observation. 5. NORINYL-1 should be used with caution in patients with a history of cerebrovascular accident. 6. In relation to breakthrough bleeding, and as in all cases of irregular bleeding *per vaginam*, nonfunctional causes should be borne in mind. In cases of undiagnosed vaginal bleeding, adequate diagnostic measures are indicated. 7. Patients with a history of psychic depression should be carefully observed and the drug discontinued if the depression recurs to a serious degree. 8. Any possible influence of prolonged NORINYL-1 therapy on pituitary, ovarian, adrenal, hepatic or uterine function awaits further study. 9. A decrease in glucose tolerance has been observed in a small percentage of patients on oral contraceptives. The mechanism of this decrease is obscure. For this reason, diabetic patients should be carefully observed while receiving NORINYL-1 therapy. 10. Because of the occasional occurrence of thrombophlebitis and pulmonary embolism in patients taking oral contraceptives, the physician should be alert to the earliest manifestations of the disease. 11. Because of the effects of estrogens on epiphyseal closure, NORINYL-1 should be used judiciously in young patients in whom bone growth is not complete. 12. The age of the patient constitutes no absolute limiting factor, although treatment with NORINYL-1 may mask the onset of the climacteric. 13. The pathologist should be advised of NORINYL-1 therapy when relevant specimens are submitted.

Side effects observed in patients receiving oral contraceptives: The following adverse reactions have been observed in patients receiving oral contraceptives: nausea, vomiting, gastrointestinal symptoms (such as abdominal cramps and bloating), breakthrough bleeding, spotting, change in menstrual flow, amenorrhea, edema, chloasma or melasma, breast changes (tenderness, enlargement and secretion), change in weigh (increase or decrease), changes in cervical erosion and cervical secretion suppression of lactation when given immediately postpartum, cholestatic jaundice, migraine, rash (allergic), rise in blood pressure in susceptible individuals, mental depression.

Although the following side effects have been reported in users of oral contraceptives, no cause and effect relationship has been established: anovulation post-treatment, premenstrual-like syndrome, changes in libido, changes in appetite, cystitis-like syndrome, headache, nervousnes dizziness, fatigue, backache, hirsutism, loss of scalp hair, erythema multiforme, erythema nodosum, hemorrhagic eruption, itching.

The following occurrences have been observed in users of oral contraceptives (a cause and effect relationship has been neither established nor disproved): thrombophlebitis, pulmonar embolism, neuro-ocular lesions.

The following laboratory results may be altered by the use of oral contraceptives: increased sulfobromophthalein and other hepatic function tests, coagulation tests (increase in prothrombin, Factors VII, VIII, IX and X), thyroid function (increase in PBI and butanol extractable protein-bound iodine and decrease in T^3 values), metyrapone test, pregnanediol determination.

norethindrone — an original steroid from
SYNTEX
LABORATORIES INC., PALO ALTO, CALIF.

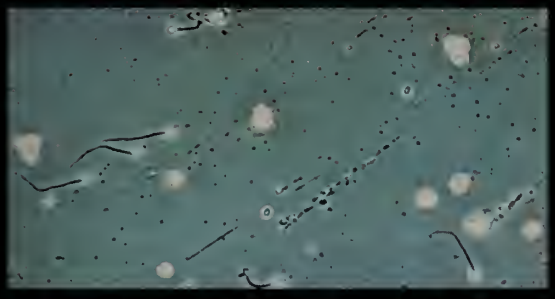
Here's why Norinyl-1 makes medical sense.

The effectiveness of Norinyl-1 as a low-dose oral contraceptive may be explained by its possible multiple action. In addition to its primary action of suppression of ovulation, Norinyl-1 may offer additional protective mechanisms... (1) creation of a cervical mucus that may be hostile to sperm penetration, and (2) development of an endometrium that may be out of phase with nidation. These effects are illustrated below.

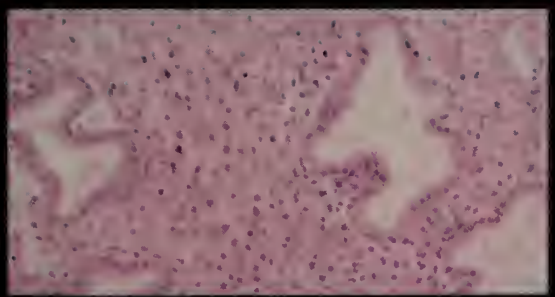
Untreated Patient



Cervical mucus at midcycle is usually thin and watery, with Spinnbarkeit (stretchability) of 15 to 20 cm.



Spermatozoa appear healthy, active, freemoving.

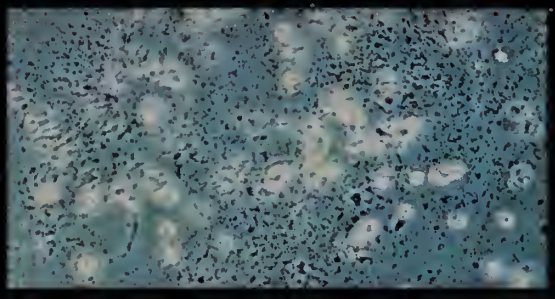


Endometrium of untreated patient is receptive to the fertilized ovum during secretory phase.

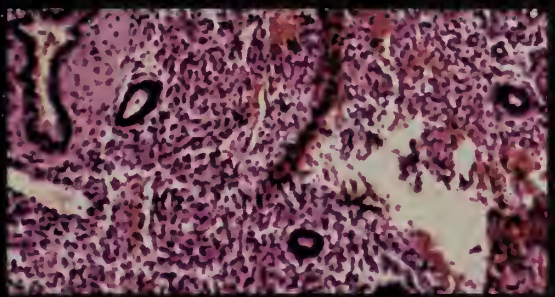
Norinyl-1 Patient



Cervical mucus at midcycle is scanty, viscous—with Spinnbarkeit of 1 cm. or less.



Immobile spermatozoa as they appear in cervical mucus taken from patient treated with Norinyl-1.



Norethindrone in Norinyl-1 accelerates secretory phase, suppresses glandular and vascular development.

Norinyl®-1
(norethindrone 1mg. & mestranol 0.05mg.) tablets

- new low dose of time-proved ingredients
- established norethindrone/mestranol ratio
- lower patient cost

Night Leg Cramps . . . Unwelcome Bedfellow

In Diabetes[1], Arthritis[2], and Peripheral Vascular Disorders[2]



now . . . specific therapy for night leg cramps

Walker

QUINAMM™

Cansistently effective, QUINAMM pravided complete relief in 94% af 200 patients studied, many af wham were severe cases refractary ta other medicatian.[3] Yaur prescription far one tablet at bedtime often cantrols painful night cramps with the initial dase . . . helps restare restful sleep.

THE NATIONAL DRUG COMPANY
DIVISION OF RICHARDSON MERRELL INC
PHILADELPHIA, PENNSYLVANIA 19144

Prescribing Informotion: Composition: Each white, beveled, compressed tablet contains: Quinine Sulfate 260 mg. and Aminophylline 195 mg. **Contraindication:** QUINAMM is contraindicated in pregnancy because of its quinine content. **Precoutions:** Aminophylline may produce intestinal cramps in some instances, and quinine may produce symptoms of cinchonism, such as tinnitus, dizziness, and gastrointestinal disturbance. Discontinue use if ringing in the ears, deafness, skin rash, or visual disturbances accur. **Dosoge:** One tablet upon retiring. Where necessary, dosage may be increased to one tablet following the evening meal and one tablet upon retiring. **Supplied:** Bottles of 100 and 500 tablets.

References: 1. Shuman, C.: Am. J. Med. Sci., 225:54, 1953. 2. Perchuk, E., et al.: Angiology, 12:102, 1961. 3. Rawls, W., et ol.: Med. Times, 87:818, 1959.

6/67 Q-706A

THE MEDICAL SOCIETY OF VIRGINIA MEMBERS' RETIREMENT PLAN

WHY YOU SHOULD BE INTERESTED IN THE KEOGH ACT

The effect of tax savings during accumulation may be simply illustrated by considering a member who is in the 40% tax bracket and wishes to invest $1,000 a year to accumulate at 6% compound interest for 20 years.

1. If he pays tax on each $1,000 before he can invest it and pays tax each year on the interest income, he will have $17,776 available at the end of 20 years. This is, in a sense, a measure of the best a man can do for himself unaided.
2. If he pays tax on each $1,000 before he can invest it but enjoys a tax free build up thereafter, he will have $23,400 available at the end of 20 years. This is a measure of the advantage of making additional voluntary contributions to an HR 10 plan.
3. Finally, if the member does not pay tax either on the amounts invested, or on the interest earnings, he will have $38,990 waiting for him at the end of 20 years. This is a measure of the advantage to be obtained by making tax deductible contributions to an approved HR 10 plan.

WHY YOU SHOULD JOIN YOUR ASSOCIATION-SPONSORED PROGRAM

1. **LOW COST**—Through the group purchasing power of the Society, sales and administrative charges are kept at a minimum.
2. **DESIGNED FOR PHYSICIANS**—Includes special features especially desirable for physicians which are not available through other plans.
3. **FLEXIBILITY**—Choice of diversified common stock or fixed return investments; opportunity to switch from one investment media to the other; contributions may be made at any time during tax year; vary amount contributed from year to year; cash or installment pay-out.
4. **SIMPLICITY**—By qualifying the Master Plan with the Internal Revenue Service, the Society makes it economical and simple for you to participate.

IF YOU WOULD LIKE FULL DETAILS ON HOW A KEOGH ACT PLAN MIGHT WORK OUT FOR YOU PERSONALLY, PLEASE COMPLETE AND RETURN THE COUPON BELOW.

- - - -



Indications: Tofrānil is recommended for the treatment of depressive states of diverse psychopathology.
Contraindications: The concomitant use of Tofrānil and monoamine oxidase inhibiting (M.A.O.I.) compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur. Potentiation of adverse effects can be serious or even fatal. An interval of at least 7 days after M.A.O.I. therapy has been discontinued should be allowed before Tofrānil may be substituted. Initial Tofrānil dosage should be low, increases should be gradual, and the patient's progress should be carefully observed.
Warning: Clinical reports have suggested that there may be a risk of teratogenesis associated with the use of this compound during the first trimester of pregnancy. Unless, in the opinion of the prescribing physician, the potential benefits outweigh the possible risks, Tofrānil should not be used during the first trimester of pregnancy.
Cardiovascular complications, including myocardial infarction and arrhythmias, have occasionally occurred in susceptible individuals. Patients with cardiovascular disease should be given the drug only under careful observation and in low dosage.
Precautions: Since suicide is always a possibility in severely depressed patients and one which may persist until significant remission occurs, such patients should be carefully supervised during early treatment with Tofrānil. Some severely depressed patients may also require hospitalization and/or concomitant electroconvulsive therapy.
Because of its anticholinergic effect, caution should be observed in prescribing Tofrānil for patients with increased intraocular pressure.
In rare instances, transient cardiac arrhythmias have occurred in hyperthyroid patients and in patients receiving thyroid medication when Tofrānil was added to the regimen.
Imipramine may block the pharmacologic activity of guanethidine and other related adrenergic neuron-blocking agents.
The drug is not recommended at the present time in patients under 12 years of age.
Adverse Reactions: Dryness of the mouth, tachycardia, constipation, disturbances of accommodation, sweating, dizziness, weight gain, urinary frequency or retention, nausea and vomiting, peripheral neuritis, mild parkinson-like syndrome, tremors, rare cases of falling in elderly patients, confusional states (with such symptoms as hallucinations and disorientation), activation of psychosis in schizophrenics and agitation (includ-

When a milestone in life is marred by depression...



Often in the mind of the lonely, widowed, depression-prone individual, she's not gaining a daughter...she's losing a son. The occasion may be marred by such symptoms of depression as feelings of sadness, incapacity, helplessness and hopelessness.

In about 3 out of 4 cases, Tofrānil relieves symptoms of primary depression.

As maintenance therapy in primary depressive illness, it helps prevent relapse.

Although toxic reactions severe enough to require discontinuation of Tofrānil are uncommon, in patients with cardiovascular disease, thyroid disorders, increased intraocular pressure, or in those receiving anticholinergics (including antiparkinsonism agents), the special precautions listed in the Prescribing Information should be carefully observed. The use of Tofrānil in patients receiving M.A.O.I.'s is contraindicated.

Tofrānil® imipramine hydrochloride
Geigy

hypomanic and manic episodes) ch may require dosage reduction l/or addition of a tranquilizer or porary discontinuation of the drug, leptiform seizures, orthostatic otension and substantial blood ssure fall in hypertensive patients, pura, transient jaundice, bone mar- depression including agranulocy- s, sensitization and skin rash luding photosensitization, eosino- lla, and mild withdrawal symptoms sudden discontinuation after pro- ged treatment with high doses. casional hormonal effects (im- ence, decreased libido, and estro- ic effects) may be observed. opine-like effects may be more nounced (e.g. paralytic ileus) in ceptible patients and in those ng anticholinergic agents (includ- antiparkinsonism drugs).
patient Adult Dosage: Initially, mg. daily, increased, if necessary, to 150 or 200 mg. Maintenance dosage may be lower, 50 to 150 mg. daily, if possible.
Geriatric and Adolescent Dosage: Initially, 30 or 40 mg. daily, which may be increased according to response and tolerance. It is usually unnecessary to exceed 100 mg. daily.
A lag in therapeutic response, lasting from a few days to a few weeks, should be expected. When dosage recommendations are already being followed, increasing the dosage does not normally shorten this latency period and may increase the incidence of adverse reactions.
Availability: Tofrānil: Round tablets of 25 and 50 mg.; triangular tablets of 10 mg. for geriatric and adolescent use; and ampuls, each containing 25 mg. in 2 cc. for I.M. administration.
(B)46-850-C
For complete details, please refer to the full Prescribing Information.

Geigy Pharmaceuticals
Division of Geigy Chemical Corporation
Ardsley, New York 10502

Chances are she'll be fever-free in less than 48 hours...



with Erythrocin®-Sulfas

ERYTHROMYCIN-
TRISULFAPYRIMIDINES,
ABBOTT

FOR RAPID CLINICAL RESPONSE

In a recent series of clinical studies, Erythrocin-Sulfas was used to treat 516 febrile cases of suspected bacterial infection. Of these, *85.5% were completely afebrile within 48 hours.*

FOR WIDE BACTERIAL COVERAGE

Among the total of 661 patients (febrile and afebrile) who could be evaluated, the *clinical cure rate* was 92.2%*

FOR OPTIMAL PATIENT ACCEPTANCE

Three forms were used in the studies, prescribed according to the age and personal preferences of individual patients:

Erythrocin® ethyl succinate-**Sulfas Granules**
erythromycin ethyl succinate-trisulfapyrimidines
for oral suspension

Erythrocin® ethyl succinate-**Sulfas Chewable**
erythromycin ethyl succinate-trisulfapyrimidines
chewable tablets

Erythrocin® stearate-**Sulfas Filmtab®**
erythromycin stearate-trisulfapyrimidines tablets

*Clinically cured included patients with complete symptomatic relief and those who had partial symptomatic relief within 72 hours with subsequent clinical cure.

803446

Please see Brief Summary on next page.

ABBOTT

BRIEF SUMMARY FOR
Erythrocin®-Sulfas

Indications For mixed infections which are more susceptible to the combination than to either agent alone. These may include susceptible gram-positive and/or gram-negative mixed infections. Not recommended in the treatment of infections expected to respond to full therapeutic doses of the antibiotic or sulfonamide alone.

Contraindications Known hypersensitivity to erythromycin or sulfonamides. Because of the possibility of kernicterus with sulfonamides, do not use in pregnancy at term, in premature or in newborn infants during first week of life.

Warnings As with other forms of sulfonamide therapy, use only after critical appraisal in patients with liver or kidney damage, urinary obstruction, or blood dyscrasias. Deaths have been reported from hypersensitivity reactions and blood dyscrasias following use of sulfonamides. Perform blood counts and liver and kidney function tests when used intermittently or for long periods.

Precautions, Side Effects Use sulfonamides with caution in patients with a history of allergy, including asthma. Mild allergic reactions (such as urticaria and other skin rashes) may occur. Serious allergic reactions have been extremely infrequent; if encountered appropriate countermeasures (e.g. epinephrine, steroids, etc.) should be administered and the drug withdrawn. Assure adequate fluid intake to prevent crystalluria and institute alkali therapy if indicated. In prolonged therapy, or when high dosage is used, maintain an alkaline urine. If oliguria occurs, discontinue therapy. Occasionally mild abdominal discomfort, nausea or vomiting may occur with erythromycin; generally improved by reduction of dosage.

If overgrowth of nonsusceptible organism occurs, withdraw the drug and institute appropriate treatment.

Adverse Reactions Sulfonamide therapy may be associated with headache, nausea, vomiting, diarrhea, hepatitis, pancreatitis, blood dyscrasias, neuropathy, drug fever, skin rash, urticaria, injection of the conjunctiva and sclera, petechiae, purpura, hematuria and crystalluria. 803446

ABBOTT



The relief received from the first Trocinate 400 mg. tablet is so prompt that the discomfort of diarrhea ceases to be a bother. May be repeated every four hours.

Upon request, a supply of Trocinate 400 mg. with literature will be sent to physicians for their personal use.

WM. P. POYTHRESS & CO., INC.
RICHMOND, VIRGINIA 23217
Manufacturers of ethical pharmaceuticals since 1856



The muscle relaxant that works before you write a prescription

Relieve painful skeletal muscle spasm in your office in minutes with a single 2 cc. injection of NORFLEX. Then, for sustained relief, write a prescription for NORFLEX tablets, 1 tablet b.i.d.

CONTRAINDICATIONS: Due to its anticholinergic action, NORFLEX should not be used in patients with glaucoma, pyloric or duodenal obstruction, stenosing peptic ulcer, prostatic hypertrophy or obstruction at the bladder neck, cardiospasm (megaesophagus) and myasthenia gravis. Use with caution in patients with tachycardia. Do not use propoxyphene (Darvon®) concurrently.

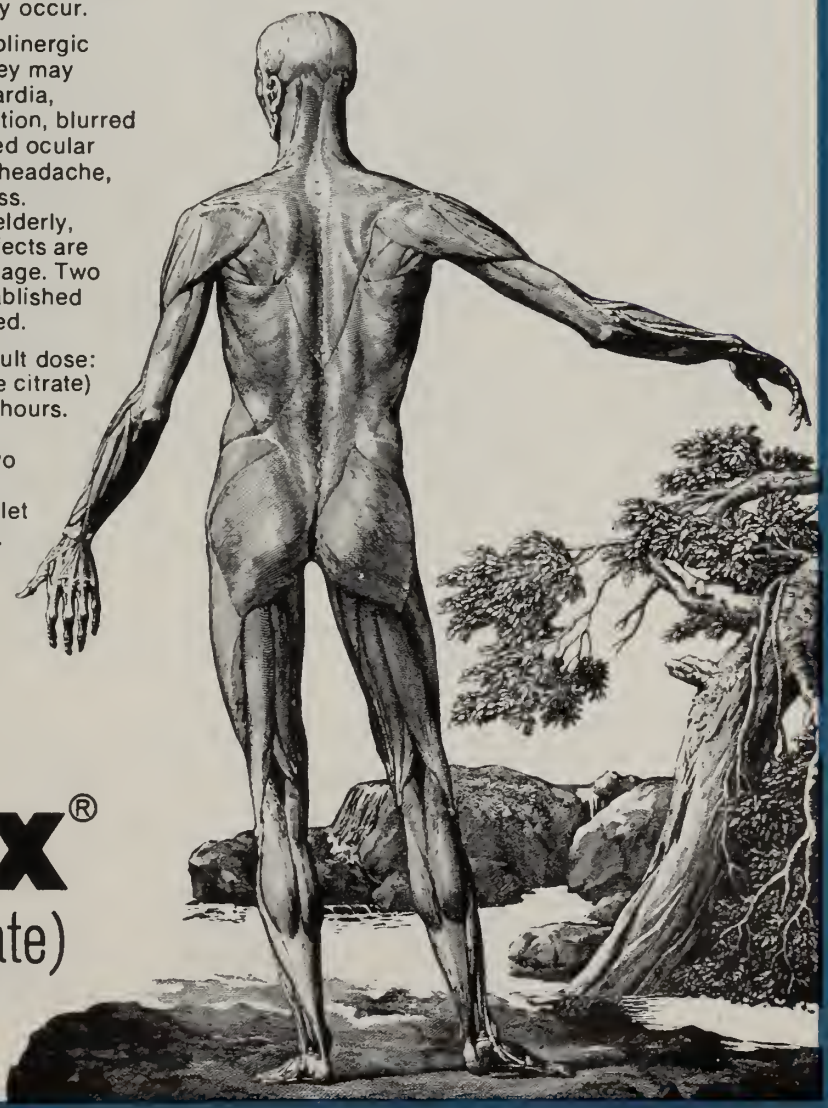
WARNING: Transient lightheadedness or dizziness following NORFLEX-INJECTABLE may occur.

SIDE EFFECTS: Due mainly to anticholinergic action and usually at high dosage. They may include dryness of the mouth, tachycardia, palpitation, urinary hesitancy or retention, blurred vision, dilatation of the pupil, increased ocular tension, weakness, nausea, vomiting, headache, dizziness, constipation, and drowsiness. Infrequently, mental confusion in the elderly, urticaria or other dermatoses. Side effects are usually eliminated by reduction in dosage. Two cases of aplastic anemia, with no established causal relationship, have been reported.

DOSAGE: **INJECTABLE** — Average adult dose: one ampul, 2 cc. (60 mg. orphenadrine citrate) I.M. or I.V. May be repeated every 12 hours. Relief may be maintained with one NORFLEX tablet b.i.d. **TABLETS** — Two tablets per day for adults, one in the morning, one in the evening. Each tablet contains 100 mg. orphenadrine citrate.

For full information, see Package Insert or P.D.R.

Riker Laboratories
Northridge, California 91324



Norflex®
(orphenadrine citrate)

mudrane®

for

- EMPHYSEMA
- ASTHMA
- CHRONIC BRONCHITIS
- BRONCHIECTASIS

The fast-disintegrating uncoated tablet gives relief in 15 minutes

Each tablet contains:

Potassium Iodide	195 mg.
Aminophylline	130 mg.
Phenobarbital, Caution: May be habit forming	21 mg.
Ephedrine HCl	16 mg.

FEDERAL LAW PROHIBITS
DISPENSING WITHOUT PRESCRIPTION

Precautions: Usual for aminophylline-ephedrine-phenobarbital. Iodides may cause nausea, long use may cause goiter. Discontinue if symptoms of iodism develop.

Iodide contraindications: tuberculosis, pregnancy.

DOSAGE

One tablet, with full glass of water, 3 or 4 times daily.

Dispensed in bottles of 100 and 1000 tablets.

MUDRANE GG—Formula, dosage and package identical to Mudrane—*except*—100 mg. glyceryl guaiacolate replaces the potassium iodide. The value of Mudrane cannot be enjoyed by a small group in which K.I. is contraindicated. Mudrane GG is prepared for this group.

MUDRANE GG ELIXIR—Four 5 cc teaspoonfuls is equivalent to one Mudrane GG tablet. Dosage adjusted to age and weight of child. Mudrane GG Elixir is for pediatric patients and those who think they cannot swallow tablets. Dispensed in pint and half gallon bottles.

WM. P. POYTHRESS & CO., INC.
RICHMOND, VIRGINIA 23217
Manufacturers of ethical pharmaceuticals since 1856



Whenever the diet is faulty, the appetite poor, or the loss of food is excessive

through vomiting or diarrhea—

Valentine's
MEAT EXTRACT

stimulates the appetite,

increases the flow of digestive juices,

provides: supplementary amounts of vitamins, minerals and soluble proteins,

extra-dietary vitamin B_{12},

protective quantities of potassium, in a palatable and readily assimilated form.

Supplied in bottles of 2 or 6 fluidounces.

DOSAGE *is 1 teaspoonful two or three times daily; two or three times this amount for potassium therapy. Dilute with two or more equal volumes of water.*

VALENTINE Company, Inc.
RICHMOND 21, VIRGINIA



"The inconvenience of a cold"

cold, NTZ® Nasal Spray provides rapid relief of symptoms. Relief starts with the first spray which s the inferior part of the common meatus. A second , a few minutes later, will shrink the turbinates to provide sinus drainage and ventilation. Dosage be repeated every three or four hours as needed, mporary relief of symptoms. NTZ is well tolerated verdosage should be avoided.

sinusitis deterrent, NTZ Nasal Spray can be used to the nasal passages open during a cold to help pre- development of acute sinusitis—or to help prevent cute condition from becoming chronic.

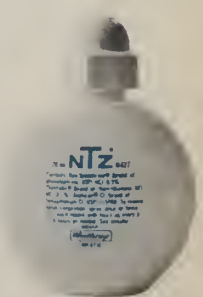
lied: NTZ Nasal Spray, plastic squeeze bottles of l.; NTZ Nasal Solution, bottles of 30 ml. (1 fl. oz.) dropper.

NTZ is more than a simple vasoconstrictor. It contains

Neo-Synephrine® (brand of phenylephrine) HCl 0.5 per cent, the major component, virtually synonymous with fast, efficient but gentle nasal vasoconstriction.

Thenfadil® (brand of thenyldiamine) HCl 0.1 per cent, topical antihistamine for reduction of rhinorrhea, sneezing or itching. It combats the allergic reactions that may occur in colds or sinusitis.

Zephiran® (brand of benzalkonium, as chloride, refined) 1:5000, antiseptic preservative and wetting agent to promote penetration and spread of the formula.



Winthrop Laboratories, New York, N.Y. 10016 Winthrop

Valium® (diazepam)

useful adjunct for the coronary patient...

When oppressive feelings and psychic tensions are severe and cooperation with the physician poor, it may be helpful to add adjunctive Valium (diazepam), 10 mg or 5 mg t.i.d., to the coronary patient's regimen. Valium (diazepam) helps to promote the needed relaxation that the patient requires to make him less preoccupied with his condition and more inclined to cooperate in, and benefit from, the total therapeutic program. Valium (diazepam) may be administered in the presence of secondary depressive symptoms.

in the hospital...

(5 mg or 10 mg t.i.d.)



convalescing...
(2 mg or 5 mg t.i.d.)

The heart patient who leaves the hospital to enter a period of slow restorative treatment is often forced to make emotionally difficult adjustments. The resulting stress of this period may again cause psychic tension to mount to potentially harmful levels. To augment your reassurance and the emotional support of his family, a 2 mg or 5 mg t.i.d. regimen of Valium (diazepam) can reduce psychic tension and thereby help the patient to accept necessary restrictions more calmly, and to adjust to his illness more realistically.

Getting back to work presents additional stresses for the cardiac patient. There are often anxiety-producing features in the job which can be more significant than the physical or intellectual demands of the work. Valium (diazepam), 2 mg or 5 mg t.i.d., may be a useful adjunct to reduce overreaction to these stresses and thus help the patient to face job situations more calmly and rationally.

back at work...
(2 mg or 5 mg t.i.d.)



Valium® (diazepam)

to help relieve psychic tension

and for the patient with tension-induced sleeplessness, remember the value of an extra tablet at bedtime

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states, moderate to severe psychoneurotic states with anxiety, apprehension or agitation alone or with depressive symptoms; somatic complaints which are concomitants of emotional factors; acute agitation due to alcohol withdrawal; muscle spasm associated with cerebral palsy and athetosis.

Contraindications: Infants, patients with history of convulsive disorders, glaucoma or known hypersensitivity to drug.

Warning: Not of value in the treatment of psychotic patients, and should not be employed in lieu of appropriate treatment.

Precautions: Limit dosage to smallest effective amount in elderly or debilitated patients (not more than 1 mg, one or two times daily initially) to preclude ataxia or oversedation, increasing gradually as needed or tolerated. As is true of all CNS-acting drugs, until correct maintenance dosage is established, advise patients against possibly hazardous procedures requiring complete mental alertness or physical coordination. Driving during therapy not recommended. In general, concurrent use with other psychotropic agents is not recommended. If such combination therapy is used, carefully consider individual pharmacologic effects—particularly with known compounds which may potentiate action of Valium (diazepam), such as phenothiazines, barbiturates, MAO inhibitors and other antidepressants. Advise patients against simultaneous ingestion of alcohol or other CNS depressants. Safe use in pregnancy not established. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Observe usual precautions in impaired renal or hepatic function. Periodic blood counts and liver function tests advisable in long-term use. Cease therapy gradually.

Side Effects: Side effects (usually dose-related) are fatigue, drowsiness and ataxia. Also reported: mild nausea, dizziness, blurred vision, diplopia, headache, incontinence, slurred speech, tremor and skin rash; paradoxical reactions (excitement, depression, stimulation, sleep disturbances, acute hyperexcited states, hallucinations); changes in EEG patterns during and after drug treatment. Abrupt cessation after prolonged overdosage may produce withdrawal symptoms (convulsions, tremor, abdominal and muscle cramps, vomiting, sweating) similar to those seen with barbiturates, meprobamate and chlordiazepoxide HCl.

Dosage—Adults: Mild to moderate psychoneurotic reactions, 2 to 5 mg b.i.d. or t.i.d.; severe psychoneurotic reactions, 5 to 10 mg t.i.d. or q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; muscle spasm with cerebral palsy or athetosis, 2 to 10 mg t.i.d. or q.i.d.

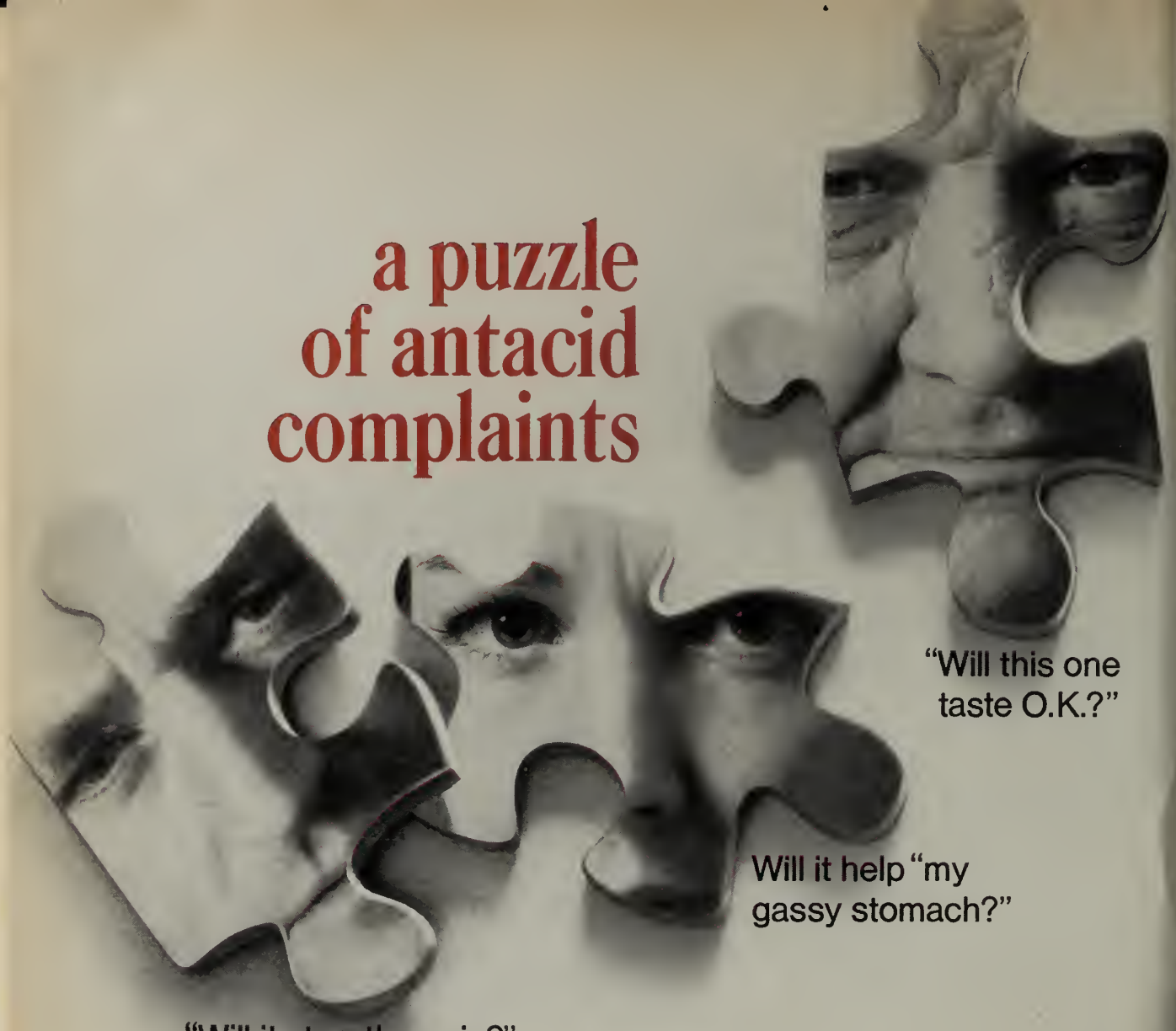
Geriatric patients: 1 or 2 mg/day initially, increase gradually as needed and tolerated. (See Precautions.)

Supplied: Valium® (diazepam) Tablets, 2 mg, 5 mg and 10 mg; bottles of 50 and 500.

ROCHE

Roche
LABORATORIES
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

a puzzle of antacid complaints



Mylanta®

aluminum and magnesium hydroxide *plus* simethicone

a solution to peptic ulcer distress

Effective neutralization—
with the two most widely prescribed antacids: aluminum and magnesium hydroxides.

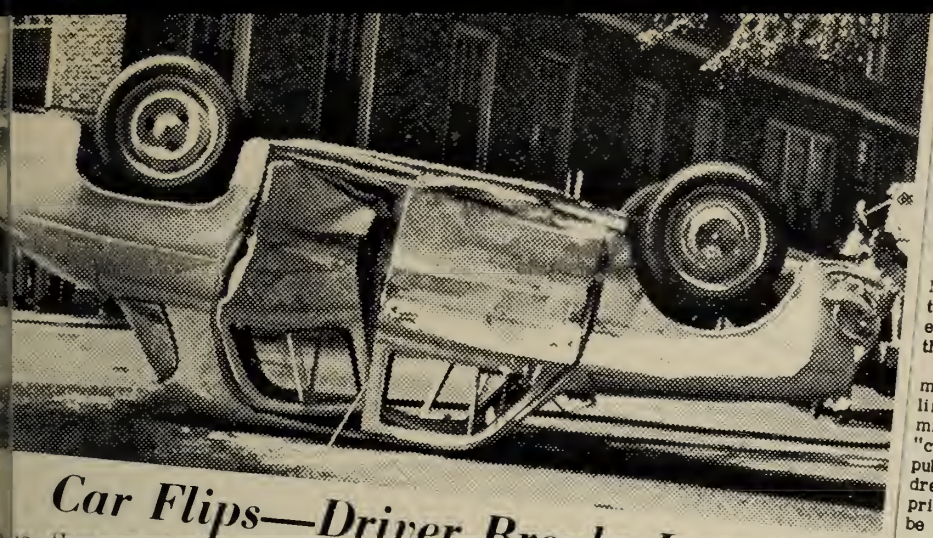
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References: 1. Hoon, J. R.: Arch. Surg. *93*:467 (Sept.) 1966.
2. Danhof, I. E., Personal communication.

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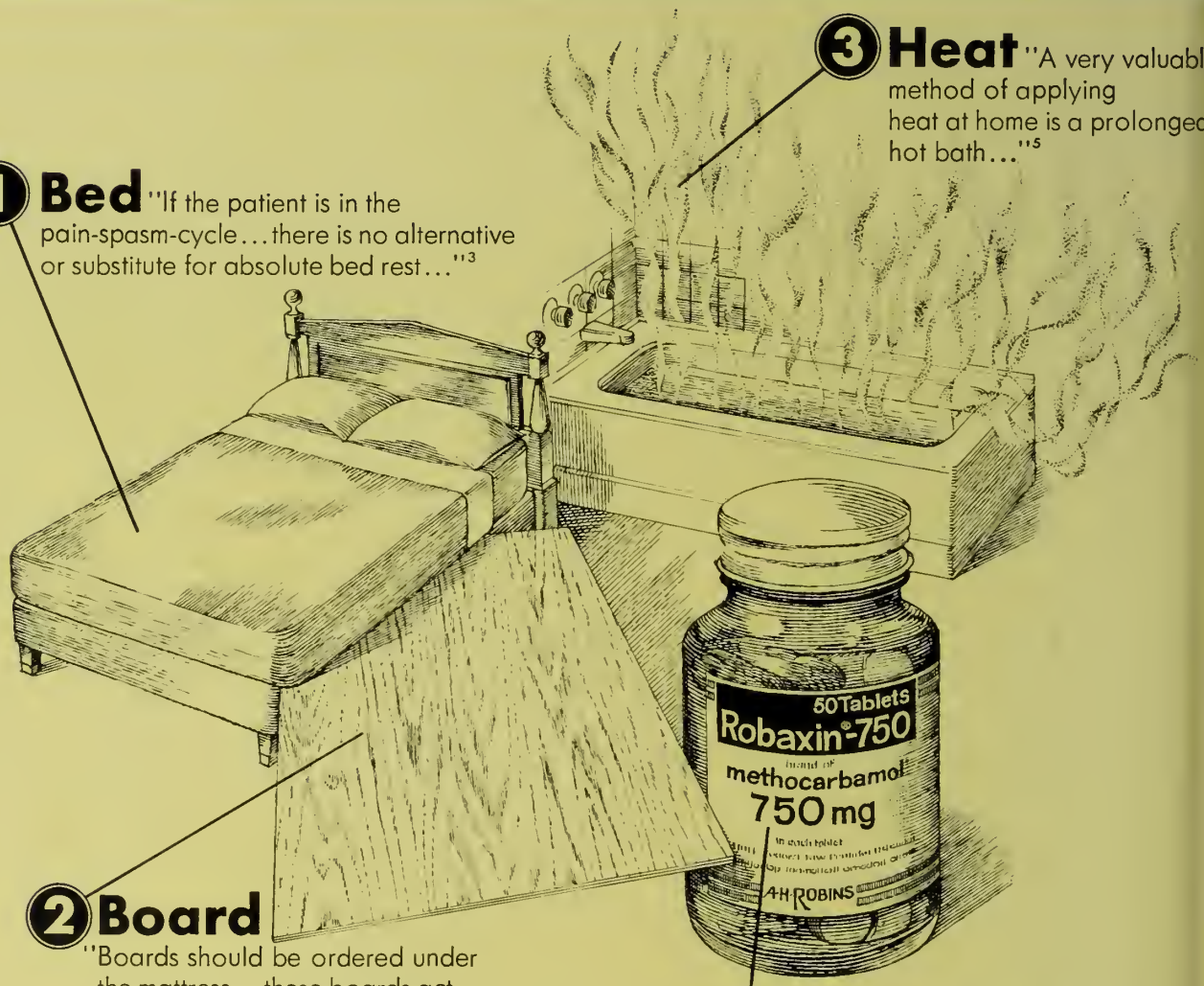
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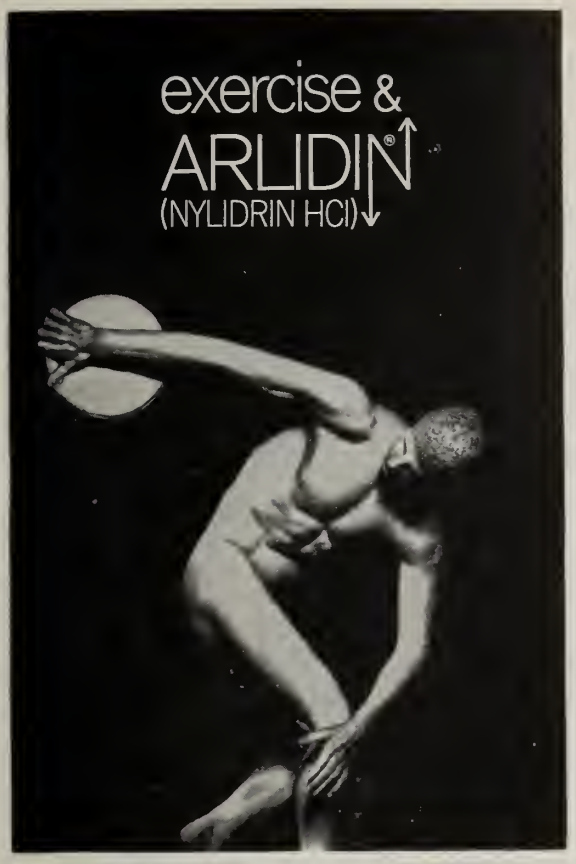


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References: (1). Godfrey, C.M.: Applied Therap. 8:950, 1966. (2). Gottschalk, L.A.: GP 33:91, 1966. (3). Rowe, M.L.: J. Occup. Med. 2:219, 1960. (4). Cozen, L.: South Dokota J. Med. 18:26, 1965. (5). Soto-Hall, R.: Med. Sc. 14:23, 1963. (6). Weiss, M. ond Weiss, S.: J. Am. Osteopath. A. 62:142, 1962. (7). Feuer, S.G., *et al.*: New York J. Med. 62:1985, 1962.

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Meprobamate: Drowsiness may occur and can be associated with ataxia; the symptom can usually be controlled by decreasing the dose, or by concomitant administration of central stimulants. Allergic or idiosyncratic reactions: maculopapular rash, acute nonthrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever, transient leukopenia. A case of fatal bullous dermatitis, following administration of meprobamate and prednisolone, has been reported. Hypersensitivity has produced fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, stomatitis, proctitis (1 case), anaphylaxis, agranulocytosis and thrombocytopenic purpura, and a fatal instance of aplastic anemia, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity, usually after excessive dosage. Impairment of visual accommodation. Massive overdosage may produce drowsiness, lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

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The Virginia
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March, 1968
Vol. 95, No. 3
Whole No. 1390

Guest Editorial . . . .

Ultrasonic Diagnosis in Obstetrics

CAN FETAL LIFE be proven at six weeks of pregnancy?

When I first read of "hearing" fetal blood flow at 10 weeks of pregnancy, I was skeptical.[1-3] Now, after six months' experience with an Ultrasonic Doppler Instrument*, I realize that the reports were conservative.

It is often possible to detect fetal blood flow with such a device at the time of the second missed menstrual period when the pregnancy is six weeks old. Reports have stated that the flow is detectable when the uterine fundus rises above the symphysis pubis. But on vaginal examination, a physician can readily lift the uterus above the pubis, pregnancy or no.

Modern electronics is therefore making us rewrite our obstetrics textbooks. Til recently the positive signs of pregnancy have been (1) hearing the fetal heart, (2) feeling fetal movements, (3) feeling fetal parts, (4) ballotting the fetus, (5) seeing fetal parts on x-ray. Lately fetal electrocardiography has added a sixth sign. Now is added a seventh —the detection of fetal blood flow and movements with ultrasound.

There is also a new sign of inevitable abortion. Heretofore, the passage of tissue, dilation of the cervix, extreme uterine bleeding, and rupture of the amniotic sac have been positive signs. Now, the disappearance of previously detected fetal flow is a fifth criterion.

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1. Bishop, Edward H. Obst. & Gynec. 28:712-713, Nov. 1966.
2. Bishop, Edward H. Am. J. Obst. & Gynec. 96:863-867, Nov. 16, 1966.
3. Johnson, Wayne L., Stegall, H. Fred, Lein, John N., and Rushmer, Robert F. Obst. & Gynec. 26:305-307, Sept 1965.

*Ultradop. The Ames Co., Division of Miles Laboratories, Inc., Elkhart, Indiana.

The Ultrasonic Doppler Instrument works on the principle that sound waves bouncing off a moving object undergo a change in frequency. Its transducer emits ultrasonic waves at six megacycles per second. When they are reflected from a moving object—a flowing column of blood, a moving limb, or surging matter in a loop of intestine —they return at a different frequency. The instrument converts this difference to sound waves, or to visible impulses if connected to an oscilloscope or electrocardiograph. The faster the reflecting surface is moving, the greater the Doppler shift.

With very little practice, one can quickly learn to detect the arterial flow of a 4 cm. embryo, the twitch of a fetal extremity, and the rush of bowel material. In later pregnancy one can find the umbilical cord, the fetal heart, and occasionally the placenta and the ascending aorta as well as other fetal arteries.

I find the machine indispensable in the management of threatened abortion. In spite of uterine bleeding, if I can detect fetal flow or movements, I know that my efforts at preserving pregnancy are worthwhile—and not directed at preserving an hydatidiform mole or delaying the passage of a blighted ovum. (Pregnancy tests, of course, indicate the presence of chorionic gonadotropin; they do not tell whether there is a living embryo attached to the chorion.) And where I have once detected fetal life, then in the face of uterine bleeding and disappearance of the flow, I can reasonably conclude that the embryo has succumbed and that procrastination is useless.

I use the Ultrasonic Doppler Instrument at every obstetrical checkup. Although I have so far not been able to discern fetal flow earlier than six weeks, I can usually do so at seven to eight weeks. To be able to prove to a mother that her fetus is viable makes the instrument well worth the investment. Once I used it to diagnose midtrimester pregnancy in an enormously obese woman with amenorrhea. I could neither hear a heartbeat nor feel the uterus. The patient, however, insisted she was feeling life, which the instrument quickly verified. I have not yet had the opportunity to test it on an ectopic pregnancy but its potential here is obvious.

It is simple to use. One merely wets the face of the transducer and places it on the abdominal skin over the uterus. In early pregnancy the uterus is generally mobile so that the transducer beam can be made to impinge on its front, top, or back side. By tipping the transducer systematically in different directions, one can bounce the ultrasonic waves off of every possible fetal location.

Because of its narrow beam, the transducer must point right at the heart in order to pick up the impulse. This helps determine fetal position. The fetal heart can often be heard over a large area. But with the ultrasonic instrument, the heart is found in only one place, namely the place where it is. This feature is especially useful in diagnosing

twins. Twin heartbeats frequently have virtually the same rates. Therefore a rate of 140 heard in the right lower quadrant may or may not come from the same heart as a rate of 144 in the left upper quadrant. With the ultrasonic instrument, a rate of 144 in the RLQ can not possibly be from the same heart as a rate of 144 in the LUQ unless the beams intersect.

It can help greatly in cases of uterine enlargement where the menstrual history is questionable, in cases of amenorrhea associated with uterine myomata, and in missed abortion. Its usefulness can extend almost to the limits of one's imagination, to the points of discovering heart disorders, monitoring the fetal heart during labor, and beyond.

Summary

Electronics technology is adding significantly to basic obstetrics.

The positive signs of pregnancy used to be:

1. Hearing the fetal heart;
2. Feeling fetal movements;
3. Feeling fetal parts;
4. Ballotting the fetus;
5. Seeing fetal parts on x-ray;
6. Fetal electrocardiography.

To this list must now be added (7): Detecting fetal life with the Ultrasonic Doppler Instrument.

The signs of inevitable abortion used to be (1) heavy uterine bleeding, (2) dilation of the cervix, (3) passage of tissue, (4) rupture of the amnion. To these must now be added (5) the disappearance of fetal arterial flow previously detected with the Ultrasonic Doppler Instrument.

The ultrasonic diagnostic instrument has a bright future. It will soon be the armamentarium of every obstetrician. The extension of its use in obstetrics and other medical fields is a certainty.

T. Stacy Lloyd, Jr., M.D.

The Pratt Clinic
1701 Fall Hill Avenue
Fredericksburg, Virginia 22401

The Clinical and Pathological Cardiac Manifestations of Rheumatoid Spondylitis

WADE L. JOHNSON, M.D.
ELAM TOONE, M.D.
Richmond, Virginia

Cardiac disease occurs in about 10% of cases of rheumatoid spondylitis. Many of these cases have a type of aortitis and endocarditis now thought to be associated with rheumatoid spondylitis.

IN 1936 MALLORY first described the pathological findings in two cases of an unusual carditis. In both of these cases the patient presented with an arthritis which was later complicated by the rapid onset of severe, fulminant, intractable cardiac decompensation. One of the patients died within three years after the onset of his carditis; the other died in less than one year. It was the appearance of this carditis that caused the discussants to diagnose both cases as rheumatic fever with mitral and aortic valve involvement. However, the singly distinct feature about these two cases stems from the fact that despite the blatantly apparent clinical evidence of mitral and aortic diastolic and systolic murmurs, the post mortem examinations in both cases failed to show any of the stigmata of mitral valve disease. In fact what was found was a histologic lesion that suggested luetic carditis: "In each case the anatomic lesion is typical of the early acute stage of luetic involvement of the heart and aorta, a lesion that for two generations has been considered pathognomonic. Yet, a considerable array of significant arguments can be marshalled against such a diagnosis." "The clinical evidence is strongly against the diagnosis of syphilis." In both cases the final diagnosis was rendered as "subacute aortitis and aortic endocarditis, etiology unknown."[21]

From this beginning and even up until the present time, there has been controversy as to the exact etiology of this type of aortitis and endocarditis. It is the purpose of this paper to set forth and describe the symptoms and signs of this cardiac entity, now considered to be associated with rheumatoid spondylitis, as they have been presented in the English speaking medical literature. It is to be remembered that many authors considered, and still do consider, rheumatoid arthritis and rheumatoid spondylitis as a single disease—the latter being considered a variant of the former. Because of this uncertainty all of those cases which were described under the name rheumatoid arthritis have been excluded.

There are 2,339 patients in this survey, all of whom were diagnosed as having rheumatoid spondylitis. Within this patient population are two who had a positive serum test for syphilis, one who had a past medical history of lues, 23 who had been diagnosed at one time as having rheumatoid arthritis, and 52 who had a past medical history of rheumatic fever. One is struck by the fact that these data will not support

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the concept that either syphilis, rheumatic fever, or rheumatoid arthritis is responsible for the cardiac pathology, since none of these entities are present with sufficient frequency to justify that type of conclusion. This survey, moreover, adds evidence to the fact that the only clinical entity present in all of the post-mortem examinations, in which this specific carditis is found, is rheumatoid spondylitis.

Clinical evidence of heart disease either by symptoms, signs or electrocardiogram is found in 238 out of the 2,339 patients. Thus, the incidence of heart involvement in this survey is 10.20%. However, it must be noted that this incidence is higher than that which is usually reported. This stems from the fact that many of the authors presented studies of a select series of patients with both the arthritis and the carditis. If these selected cases are excluded from this consideration, then the incidence of heart involvement in rheumatoid spondylitis is seen to be 7.92% in this series.

The joint disease was found to have an onset at the average age of 28.19 years; and the cardiac involvement began about 15.4 years later at the average age of 43. Death occurred in these patients at an average of 4.88 years after the onset of the heart disease and 20.28 years after the onset of the joint disease at the average age of 48. Not all of the series reported the age of death, so consequently, the figures for the total duration of rheumatoid spondylitis and the heart disease are taken from a smaller segment of the patient population.

Out of the total 2,339 patients, 238 showed objective evidence of heart disease. The diagnosis of congestive heart failure was reported in 38 patients (16%); angina-like pain was complained of in 13 patients (5.46%); and 14 (5.88%) experienced palpitations.

Physical examination indicated the diagnosis of acute pulmonary edema in nine cases (3.78%) while dependent edema was present in eight (3.36%). Excluding the auscultative evidence which will be presented later, the physical signs revealed the peripheral manifestations of aortic insufficiency in 30 patients (12.6%) while cardiomegaly was demonstrated in 38 (16.0%). Cardiac findings of some type were reported in almost all of the 238 patients so that a comparison with the total cardiac population is much more meaningful. The average reported blood pressure was 157/58 mm Hg, while the average pulse pressure was 97.3 mm Hg. The greatest two pulse pressures which were reported were present in one patient[3] who had a BP=140/0 hence a pulse pressure of 140 mm Hg; and in a second[14] who had a BP=230/70 with a pulse pressure of 160 mm Hg. The smallest pulse pressure was 55 mm Hg which was recorded in one of the two patients originally described by Mallory, both of whom had the murmur of aortic insufficiency.

Auscultation of the heart revealed the murmur of aortic insufficiency in 112 patients for an incidence of 47.2%; while the aortic stenosis murmur was present in 30 (12.6%). The mitral insufficiency murmur was present in 44 or 18.5%, while only 19 (8.0%) had the murmur of mitral stenosis. The Austin Flint type murmur was thought to be present in 10 patients, 4.2% of the cardiac cases; and non-specific murmurs were reported in 18 (7.57%). A ventricular diastolic gallop rhythm was present in six instances (2.5%).

It is apparent from these data that the major physical finding at the cardiac examination is the murmur of aortic regurgitation. The question would naturally arise as to the comparison of the frequency of rheumatic fever among those patients with the aortic and mitral insufficiency murmurs. It should be recalled that there were 52 patients who gave a past medical history of rheumatic fever. In one series (13) in which there were 45 cases of a past medical history positive for rheumatic fever, only eight patients showed the aortic insufficiency murmur and only two patients had the

murmur of mitral insufficiency out of a total of 352 with diagnosed rheumatoid spondylitis (see Chart A). The question now arises as to whether the patients with the murmurs were the same patients who had the history of having had rheumatic fever. Or, stated another way—did rheumatic fever produce these insufficiency murmurs? Schielder et al.[1] do not believe that rheumatic fever played a role in this one patient's heart disease since post mortem examination revealed no stigmata of rheumatic fever. Only the cardiac changes associated with rheumatoid spondylitis were found. Bernstein[13] takes a different view. She believes that rheumatic fever does play a dominant role. Of the 16 patients who had clinically manifest heart disease, 11 had a past medical history of rheumatic fever. Out of the 47 pathological electrocardiograms, she reported, 10 of these were in patients with a history of rheumatic fever, and she further states "in 42 patients . . . the heart affection was, with extreme probability, of rheumatic etiology." "It is, therefore, beyond doubt that the acute rheumatism syndrome plays a very important role in the pathogenesis of the cardiac complications." The criticism that may be made about these statements centers about the fact that she does not validate them with post mortem studies. Davidson et al.[15] found one case of definite rheumatic fever, and this occurred in the one patient who had isolated mitral stenosis found by clinical examination. However, this was in a clinical series and no post mortem examination was performed. Blumberg et al.[19] admitted that rheumatic fever was present in the history of the majority of patients with valvular disease but declined to speculate as to whether or not this played a role in the etiology of the valvular disease since no autopsy examinations were performed. It seems more logical in the absence of objective evidence to the contrary to assume that rheumatic fever did not play a prominent role.

Among the 238 patients who had evidence of cardiac disease, electrocardiographic examination revealed 90 (37.8%) tracings that were read as first degree heart block. Second degree heart block was found in five patients (2.10%); and third degree heart block was found in nine (3.78%). At least one patient[20] demonstrated a permanent progression in the degree of his heart block, going from second degree to total heart block. There were 15 cases (6.30%) in which the EKG was read as showing various other types of arrhythmias among which were atrial fibrillation, sinus tachycardia, and non-specific S-T wave changes. Right bundle branch block was reported in three patients (1.26%); and 10 (4.22%) showed left bundle branch block. The Wenckebach phenomenon was reported three times (1.26%). And finally, left ventricular strain or hypertrophy was the EKG diagnosis in 20 patients (8.41%).

In the cardiac population of 238 patients, the roentgenographic diagnosis of left ventricular hypertrophy was rendered in 42 cases (17.64%). Dilatation of the aorta was

CHART A

Series	Total Patients with Rheumatoid Spondylitis	Past Medical Diagnosis of Rheumatic Fever	Cardic Murmurs			
			AI	AS	MI	MS
(1)	5	1	5	5	5	5
(13)	352	45	8	3	2	4
(15)	1000	1	14	5	5	2
(19)	142	5	4	5	5	1
		52				

the x-ray diagnosis in eight patients (3.3%).

In the literature surveyed, two patients were found in whom aortic valvular prostheses were employed. These two patients were among the five case histories that Schielder et al.[1] reported in 1956. The first patient had a Hufnagel plastic aortic prosthesis implanted in the first part of his descending aorta because of his "severe aortic insufficiency". At the first month post-operative check-up the patient showed considerable symptomatic improvement with a definite decrease in heart size. However, twenty months post-operatively the patient died suddenly. The cause of death was not recorded. The typical changes of rheumatoid spondylitis carditis were found in his heart. The second patient underwent cardiac surgery for the implantation of a Hufnagel plastic aortic valve prosthesis in the first part of his descending aorta. Unfortunately there was no post-operative follow-up reported.

The general consensus seems to be that the carditis of rheumatoid spondylitis is benign. In view of the fact that the herein reported dates show that the carditis appeared 15.4 years after the onset of the arthritic changes and that the carditis lasted an average of 4.88 years before death, this opinion is certainly open to question. The opposite would seem to be the only logical conclusion in light of the data presented.

It is appropriate to mention, at this point, that several other conditions were reported with a higher than chance frequency in the 2,339 rheumatoid spondylitis patients. There were 12 cases of pericarditis, 16 of uveitus, 35 of urethritis, 47 of anemia, and 157 of iritis. These entities are not present in sufficient number to warrant any definite conclusion as to their relationship to the carditis of rheumatoid spondylitis.

Out of a total of 91 deaths reported in the literature post mortem examinations were done on 30 patients thought to have spondylitic carditis. In all of the cases the gross examination of the hearts revealed cardiomegaly with either hypertrophy, dilatation, or both. In 20 (66%) of the 30 hearts the aortic valve and aortic valvular ring were dilated. The commissures of the aortic valve were completely separated in 22 (73.4%) of the 30 cases; and in a few instances one or two of the commissures showed fusion for a short distance.[3] There was at least one case[20] in which two out of the three commissures of the aortic valve showed partial fusion. Thickened, retracted, rolled, shortened cusps were found at the aortic valve in 25 hearts (84.4%). Dilatation of the proximal aorta was found in five (20%). There were 17 hearts (56.6%) which showed intimal plaques in the sinus of Valsalva beginning at the aortic cusp and extending into the sinus and then up the proximal aorta a short distance. These intimal plaques:

> "centered above each valvular commissure and blended with the valvular lesions. They extended into the sinus of Valsalva and extended from the commissure 2.5 cm up the ascending aorta. Occasionally discrete plaques up to 1 cm in size were present in the proximal portion of the ascending aorta. . . ."
>
> "The appearance of the plaques varied, depending on the duration, activity, intensity of the lesion, from a pinkish red, coarsely granular 'pannus-like' layer to a pearly gray, smooth, glossy 'sugar icing' type of intimal thickening or depressed regions of egg shell calcification." [3]

There were two cases[10,14] in which these plaques were found in the arch of the aorta and one case[14] in which they were found down to the level of the renal artery. In 46 out of the 50 cases (92%) examined, no gross abnormalities were found at the mitral valve.

It is the microscopic picture that comprises the most unique and distinct feature of the heart involvement found to be associated with rheumatoid spondylitis. The aortic cusps showed:

"only slight vascularization and various

degrees of thickening with dense collagenous fibers. The fibrotic thickening, more dense at the base of the cusps, extended into the adjacent myocardium with linear and whorle formations." [15]

". . . whorled masses of hyalinized collagenous tissue (were revealed) in the free edges, base, and annulus of the aortic valves."

". . . at the base of the valves marked fibrosis with thickening extended into the annulus." [1]

In these areas of valvular cusp fibrosis and thickening and the regions of the myocardium adjacent to the aortic valve ring, a distinct peculiarity of the arterioles was noted. Davidson et al.[15] reported: "Small vessels in these areas evidenced moderate medial hypertrophy and intimal hyperplasia and hypertrophy." Weed et al.[10] wrote: "Arterioles in the regions of fibrosis had strikingly thickened walls and narrowed lumens." Graham et al.[12] stated: "Slight fibromuscular thickening of the intramural arterial branches in the myocardium was noted." As stated above, the aortic valvular ring was affected both by the fibrosis and the arteriolar change. "In the aortic valvular ring there was focal collagen fragmentation, fibrosis and some infiltration with lymphocytes and myocytes." [3] "There were few tiny vessels in this hyalinized tissue and scattered lymphocytes surrounding these at the vascular base." [1]

"The chronic aortic valvular lesions differed from those of rheumatic fever in that the chronic inflammatory changes occurred primarily at the bases rather than at the free margins of the cusps . . . also they lacked the marked fibrotic fusion and valvular stenosis which often occur in association with rheumatic fever." [15]

The aorta also showed changes which were unique, but also a source of great confusion for etiological thinking. The intimal plaques were "the result of subendothelial connective tissue proliferation, and increase in mucinous ground substance, with little or no associated inflammatory cellular infiltration." [3]

". . . the aortic media in the region of the aortic valve ring contained scattered foci of collagen and elastic tissue destruction with vascularization, areas of round cell infiltration, and occasional polymorphonuclear neutrophils." [12]

The proximal portions of the ascending aorta showed:

"irregular focal destruction of the media with necrosis of muscle fibers, fragmentation of elastic lamellae and ingrowth of vascular granulation tissue containing varying numbers of lymphocytes, small mononuclear wandering cells, and neutrophils. The inflammatory cells tended to surround the penetrating vessels. No multinucleated giant cells of the Langhans or foreign body type were noted." [3]

The pathology responsible for these destructive changes in the media centers about vascular changes present in the adventitia of the aorta. "The adventitia in the region of the intimal changes tended to be fibrotic and fused to the media." [3]

"The adventitia showed varying degrees of perivascular round cell infiltration, connective tissue proliferation, mucinous edema, and fibrosis. The most striking change, however, was a marked fibromuscular thickening of the vasa vasorium with complete obliteration of the lumen in some instances." [3]

". . . most of the small arteries at the base of the aorta exhibited a marked degree of fibrosis, mural thickening with consequent compromise of the vascular lumen. Others were surrounded by prominent focal accumulations of lymphocytes and plasma cells." [20]

The change that has taken place in the adventitia is an obliterative endarteritis of the vasa vasorium with apparent subsequent embarrassment of the blood supply to the media resulting in tissue destruction and then repair by fibrosis. The same is appar-

ently true of the tissue destruction in the aortic cusps and aortic valvular ring and adjacent myocardium.

> "The histologic changes present . . . were more characteristic of those seen in syphilis than in rheumatic fever. The tunica externa showed endarteritis obliterans of the vasa vasorium and perivascular infiltration composed principally of lymphocytes and plasma cells. There was focal destruction of the elastica of the media with replacement by linear scars. The intima was thickened with focal inflammatory areas composed of polymorphonuclear cells, lymphocytes and plasma cells." [2]

Mallory described the histological picture as follows and then admitted the confusion which set the stage for the subsequent years of etiological controversy when he stated in 1936:

> "Histologically the intima of the involved areas shows a marked overgrowth of fibrous tissue with little or no inflammatory infiltration. The media under these areas is invariably highly vascularized with foci of absorption of muscle cells and elastic lamellae. The penetrating vessels are often surrounded by cuffs of lymphocytes. In the adventitia the perivascular inflammatory infiltration is very marked."

Yet, nowhere in the literature has a report appeared of *Treponema pallidum* ever being demonstrated in any of these lesions; and, for that matter, the vast majority of these patients have persistently demonstrated negative serological tests for syphilis. Aschoff bodies, onion skin lesions, and rheumatoid nodules have not been demonstrated in these hearts.

In this present series 23 out of the 30 aortic valves (76.6%) showed an increase in connective tissue. Endarteritis obliterans with subsequent collagenous fragmentation and fibrotic repair was present in the aortic valvular ring and the adjacent myocardium in 19 out of the 30 cases (63.4%). The vessels in the region of the mitral valve also showed endarteritis obliterans in 19 instances (63.4%). Yet, 46 out of the total 50 autopsies showed mitral valves that were normal, with perhaps some increase in connective tissue reported in one series.[15] The intima of the aorta was reported thickened in 19 cases (63.4%) while the media was described as destroyed in 15 (50%). However, there were 19 aortas (63.4%) in which the media was described as showing elastic lamellar and muscle cell fragmentation. Endarteritis obliterans was present in the adventitia of 19 aortas (63.4%). Complete obliteration of the lumen of the vasa vasorum was reported in three, partial obliteration was reported in three, and a non-specified compromise of the vascular lumen was reported in 13 patients. The adventitia showed an increase in connective tissue in 18 aortas (60%) while mucinous edema was described in the adventitia of nine (30%).

At this point it is both logical and appropriate to answer the question of whether or not the pathological post mortem findings will explain the major cardiac manifestations of rheumatoid spondylitis. The aortic insufficiency that seems to be so characteristic of this disorder is easily explained when one remembers that the aortic valvular ring has been destroyed in part and then repaired by fibrosis. This has occurred to the extent that at least 66% of the hearts had dilated aortic valvular rings. There would come a point in the process of dilating that the valvular cusps would no longer approximate and aortic insufficiency would result.

> "If a chamber of the heart becomes dilated, the leaflets of one of its valves may fail to close the orifice completely and relative insufficiency results. Or, if the chamber distal to the orifice dilates and the chamber proximal to its remains normal, the valve orifice will be relatively normal in comparison to the dilated chamber and relative stenosis will be the result." [23]

The aortic stenotic murmur could be considered a relative stenotic murmur. This would occur if the aorta above the aortic valve dilated until a point was reached such that the supravalvular aortic dilation was great enough to render the dilated aortic valvular ring relatively small by comparison. At this point the flow would be through a valvular orifice that, even though dilated, was small relative to the distal chamber, the aorta in this case. Medial destruction—fragmentation of the elastic lamellae and muscular cells—was present in 63.4% of the aortas examined and gross dilatation of the aorta was present in 20%. The presence of the fragmented medial elements would suggest that under pressure the aorta would readily dilate; and the presence of the gross aortic dilatation indicates that the dilatation was a fact. It therefore becomes highly probable that the aortic stenotic murmur is a relative murmur.

The murmur of mitral insufficiency may be explained in much the same manner. All of the hearts showed cardiomegaly with left ventricular hypertrophy or dilatation or both. There would come a time when the chamber had dilated to such an extent that the mitral valvular leaflets would not approximate in systole; and the murmur of mitral insufficiency would be produced.

In view of the fact that 92% of all of the hearts examined had no gross abnormality of the mitral valve, the presence of the murmur of mitral stenosis can probably best be explained on the basis of an Austin Flint murmur.

> "The term (Austin Flint murmur) today is generally used to designate the diastolic rumbling murmur heard at the apex, in a patient who has an aortic insufficiency, not due to organic mitral stenosis." [23]

> "Statistically the Flint murmur may be diagnosed when other clinical features are absolutely typical of moderate or severe aortic regurgitation and there are no other auscultatory signs of mitral stenosis or mitral calcifications by x-ray. It has been stated that the only definitive diagnosis of the Flint murmur is at the autopsy table or in the cardiac catheterization laboratory." [24]

A combination of factors is thought to be responsible for this murmur and several theories have been brought forth.

> "One of the theories suggests that with sufficient aortic insufficiency, the aortic leaf of the mitral valve is displaced into such a position that it produces a relative mitral stenosis." [24]

> "Another possible mechanism is that a relative mitral stenosis results from the excessive enlargement of the left ventricle as compared to the left atrium." [25]

The following points strongly suggest that the mitral stenosis is actually the Austin Flint murmur in this series: (1) The lack of abnormalities on the mitral valve, (2) The presence of aortic insufficiency, (3) The presence of left ventricular hypertrophy, and finally, (4) The absence of any physical findings that could be called sequelae of mitral stenosis especially the absence of dilation of the left atrium. It is suggested, therefore, that the murmurs of mitral stenosis reported here were in reality the Austin Flint rumble.

The conduction defects, which had a frequency greater than 59% among the cardiac patients, are probably due to the close proximity of the conduction system to the aortic valve and an extension of the endarteritis among the vessels supplying the bundle of His, the A-V bundle, and the bundle bronchus. Ischemic necrosis destroying part or all of the conduction system and repair by fibrosis with scar formation are due to endarteritis obliterans has been shown in only two patients. Because of the rarity of this finding, both of these pathological reports will be presented.

> "Step reactions of the conduction system by the method of Lew et al. showed continuity of the fibrotic and endarteritic process from the base of the aortic valve to the apex of the muscular septum. The continuity of the proximal right bundle

branch, fascicles of the left bundle branch, and the horizontal portion of the bundle of His appeared to be intact. The penetrating portion of A-V bundle, however, was almost completely replaced by fibrotic tissue continuous with that present in the aortic root. A few recognizable striated muscle fibers remained in this region embedded in the fibrous tissue, evidently remnants of the common bundle." [10]

The above patient had the onset of his carditis with aortic insufficiency and left sided congestion failure. In the space of three years he acquired a conduction defect which for a year was described as an atrial flutter with 2:1 or 3:1 block which varied between the two. Three years after the onset of the carditis, the patient had complete heart block and died because of pulmonary embolism. In the second reported case:

"Dissection of the A-V node, the bundle of His, and the bundle branches was carried out according to the methods of Lew et al. Serial sections showed complete destruction of the horizontal conduction bundles with replacement by fibrous scar tissue intermixed with prominant accumulations of lymphocytes and plasma cells generally orientated about small blood vessels. These vascular lesions punctuated the more superior portions of the membranous interventricular septum so that there was continuous linear distribution of foci of chronic inflammatory cells from the most inferior portions of the membranous septum (The region of the conduction fibers) to the base of the aortic cusps, and then extending further into the root of the aorta." [26]

"Because of the morphological proximity of the aortic ring and the horizontal conduction bundle, diseases involving the root of the aorta should not unexpectedly have conduction manifestations." [20]

Summary

Cardiac disease was found in 238 (10.2%) of 2339 cases of rheumatoid spondylitis reviewed in the English literature between 1950 and 1966.

Of these 238 cases, 52 gave a history of having had rheumatic fever; 23 showed evidence of peripheral rheumatoid arthritis; and two had positive tests for syphilis, one of whom gave a past history of having had the disease.

Clinically the heart involvement is characterized by cardiomegaly, aortic insufficiency, conduction defects, anginal pain, and congestive heart failure. Physical findings of mitral valve disease are rare.

The pathological features are distinctive and more closely resemble the changes seen in syphilis than in rheumatic fever. The most characteristic pathology occurs in the root of the aorta. The intima is thickened, plaque formation present, and often cellular infiltration. The elastic tissue of the media is disrupted, fragmented and replaced with fibrous tissue which is often quite cellular. The adventitia change is characterized by the striking development of an endarteritis obliterans. The aortic ring is dilated, the valve incompetent, and the valve leaflets rolled and thickened at the margins and are usually not fused but separated. The mitral valve is not involved.

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ANNUAL MEETING

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The Surgical Management of Iliofemoral Venous Thrombosis

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Iliofemoral venous thrombectomy in the management of deep venous thrombosis has much to offer for relief of early morbidity, prevention of pulmonary embolism, and prophylaxis for the postphlebitic syndrome.

THE ENLARGING EXPERIENCE with iliofemoral venous thrombectomy should assure this operation a place in the management of deep venous thrombosis. Enthusiasm is lacking, however, because of relatively satisfactory early results of conservative management and fear of producing pulmonary embolism by operative manipulation. However, the patient with iliofemoral venous thrombosis must face the risk of late chronic deep venous insufficiency. The incidence of this sequel varies from 50 percent to 90 percent in untreated cases[1,11,16] and may be as high as 60 percent in patients treated with anticoagulants alone.[9] Furthermore, the incidence of intraoperative pulmonary emboli is certainly less than 3 percent,[18] and only two fatalities have been reported.[3,12] It would therefore appear that both objections have little basis in fact.

By comparing results of surgery with conservative treatment in similar cases, we attempted, two years ago, to demonstrate three advantages of iliofemoral venous thrombectomy: relief of early morbidity, prevention of pulmonary embolism, and prophylaxis for postphlebitic syndrome.[18] (Table I)

Duration of hospitalization was used as one criterion of relief of early morbidity, but immediate relief of pain and reduction of edema in surgical patients was even more impressive in evaluating this factor. There was one questionable episode of non-fatal pulmonary embolism in the surgical group, and the one death in the medical group was from a massive pulmonary embolus. It is apparent that, over a short term, the control of venous insufficiency was more successful among the patients treated surgically. From these results it was concluded that thrombectomy is superior to conservative management in the relief of early morbidity, is at least as successful in the prevention of pulmonary embolism, and may perhaps be more effective in alleviating the long-term sequellae. Our medical colleagues were apparently convinced and the surgical series has almost tripled during the past two years.

Indications for Surgery

The basic indication for iliofemoral venous thrombectomy is the diagnosis of either phlegmasia alba or phlegmasia cerulea dolens. Restoration of venous flow results in the rapid resolution of edema and relief of pain. Removal of the thrombus obviates the risk of pulmonary embolism, and salvaging the venous valves aborts the postphlebitic syndrome at its inception.

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Table II outlines the absolute and relative contraindications to this procedure. Surgical therapy offers little advantage to the patient with established deep venous insufficiency secondary to a previous episode of thrombophlebitis. Furthermore, the presence of an associated lethal or incapacitating disease should dampen surgical enthusiasm.

Involvement of the inferior vena cava is heralded by bilateral massive lower extremity edema. In this circumstance the conventional approach to iliofemoral venous thrombectomy from the groin is contraindicated. With proximal caval control, however, this situation may still be surgically corrected.

The duration of thrombosis compatible with surgical removal is poorly defined, and some believe that all patients should be surgically treated regardless of the duration of the thrombosis.[2,13,14,19] Our experience indicates, however, that a clot in place for more than seven to ten days is too well organized to allow for complete surgical success. This relative contraindication does not apply to phlegmasia cerulea dolens, where every effort should be made to salvage the afflicted limb from venous gangrene.

If a pulmonary embolus has occurred prior to surgical intervention, discretion would seem to dictate that the inferior vena cava be interrupted proximally before attempting a thrombectomy. However, some believe that thrombectomy alone is sufficient therapy even in this circumstance.[5,14]

For anatomic reasons, supported by clinical experience, venous thrombosis originating in the veins of the calf cannot be completely extracted. Fortunately phlegmasia dolens originates in the iliofemoral region in 60 to 75 percent of afflicted patients.[4,10,15,18] Furthermore, despite incomplete removal of distal thrombus, the preservation of valves in the superficial femoral and popliteal veins may be of value. Thus these patients may be surgically treated with the expectation of satisfactory, but not perfect, results.

Finally, preoperative phlebography as a diagnostic procedure is generally unnecessary because the clinical diagnosis of iliofemoral venous thrombosis is so apparent that further confirmation is superfluous. Thus we have not routinely employed this technic and, as a result, we have performed two negative explorations, neither of which was detrimental to the patients.

TABLE I

Comparison of Medical and Surgical Management

Number of Cases	Mean Age (years)	Side		Mean Duration of Hospital Stay (days)	Results of Treatment				Mean Duration of Follow-up (months)
		Left	Right		Excellent	Satisfactory	Failure	Death	
Medical 16	49	14	2	20	2 (12%)	6 (38%)	7 (44%)	1 (6%)	16
Surgical 18	52	15	3	14	7 (39%)	7 (39%)	3 (16%)	1 (6%)	17

Table II

Indications and Contraindications for Iliofemoral Venous Thrombectomy

Indications

Phlegmasia cerulea dolens—*no time limit*
Phlegmasia alba dolens

Absolute Contraindications

Terminal or critically ill patient
Prior deep venous insufficiency

Relative Contraindications

Bilateral Involvement (caval involvement)
Duration of symptoms greater than seven days
Preoperative pulmonary embolus—qualified
Process initiated in calf veins

Operative Technic

The technic of iliofemoral venous thrombectomy is appealing and universally applicable in its simplicity. To facilitate dissection, anticoagulants are not administered prior to surgery. Heparin is given intravenously after the femoral vein is isolated and just prior to venotomy. Because of the documented risk of dislodging an embolus during surgical preparations, cleansing and draping is performed gently. The use of local anesthesia reduces the operative risk and enables the patient to cooperate during surgery. An alert patient can assist in the removal of proximal clot by straining. This Valsalva maneuver also increases venous pressure, creating a gradient through the open venotomy and further diminishing the risk of pulmonary embolism. A vertical, rather than the usual oblique, incision over the femoral triangle is superior because it transects fewer engorged lymphatic channels, thus reducing postoperative wound complications. In dissecting free the common femoral vein, the surgeon must carefully avoid injuring the deep femoral branches which may be multiple and are hidden posteriorly.

Of the many technics for extracting the thrombus, only a few are of value. After the venotomy, the first step is to have the patient strain down while the surgeon applies manual pressure to the lower abdomen. This alone will frequently extrude the major portion of the proximal clot. The most useful tool for removal of the proximal thrombus is the Fogarty catheter.[7,8] If, as is occasionally necessary, the inferior vena cava has been occluded proximally with a clip, care must be taken to avoid disrupting this clip by inserting the balloon catheter too far proximally. This instrument is less effective distally, where its progress is often impeded by valves which may be damaged by forceful manipulation. Distal thrombus is best removed by vigorous, orderly massage, progressing from the ankle to the groin. For this reason, the entire extremity should be included in the operative field.

Because it is most important to achieve good bidirectional flow during surgery, it is well to remember that there is a valve above the inguinal ligament in 81 percent of cases.[6] This valve must be made temporarily incompetent before proximal patency can be assessed. Blood loss during this procedure is insidious, but quite significant, and blood for transfusion must be available. Finally, the venotomy is closed with a continuous vascular suture and the incision is closed with drainage. Tissue drains are necessary both because of the lymphatics severed during surgery and the postoperative use of anticoagulants.

Postoperative Care

The primary goal in the postoperative period is the prevention of recurrent thrombosis. For this reason patients are maintained on intravenous Heparin and clinical Dextran for five days, followed by administration of oral anticoagulants for six weeks. Postoperative antibiotics are used only for specific indications. The affected limb is elevated and given full-length elastic support which is maintained for one year whether or not there are residual signs of deep venous insufficiency. Finally, because venous stasis is promoted by inactivity, early ambulation is mandatory. The danger of dislodging emboli is remote after the vigorous efforts already directed to this end, and active exercise, massage or ambulation are indicated within 12 to 24 hours of surgery.

Material and Results

Forty-seven patients were treated by iliofemoral venous thrombectomy. (Table III) Two negative explorations left 45 determinant cases. Age and sex distribution, side of involvement and etiologic factors are consistent with reported features of this disease regardless of method of management.

The operative removal of a large thrombus is immediately gratifying. (Fig. 1) The almost immediate resolution of edema and relief of pain are dramatic, although evaluation is necessarily subjective. This group of patients is too small to evaluate success in preventing pulmonary embolism; however, no intraoperative or early postoperative pulmonary emboli have occurred. There were two late emboli, one fatal. Both occurred more than three months after thrombectomy, and in one instance the embolus followed another surgical procedure.

TABLE III

PATIENTS TREATED WITH ILIOFEMORAL VENOUS THROMBECTOMY

Number of Cases: 47
Negative exploration: 2
Determinant Cases: 45

Side Involved:
Right: 12
Left: 33

Sex:
Female: 18
Male: 29

Average Age: 46 years (20 years-83 years)

Etiology:		
Surgical	14	Associated with Oral Contraceptives: 3
Medical:	17	Associated with Pregnancy: 4
Spontaneous:	6	
Trauma:	7	

Average duration of symptoms: 58 hours
Range: 2 hours to 16 days

Results relative to the control of deep venous insufficiency (Table IV) have been evaluated largely on clinical grounds because phlebographic studies do not correlate well with the clinical outcome.[5,17,19] Asymptomatic patients who do not require elastic support are considered excellent results. Minimal symptoms of pedal edema or aching pain, or only intermittent need for elastic hose is considered satisfactory. All other results are classified as failures. On this basis, of the entire group of patients, 47 percent have achieved an excellent result, 29 percent are satisfactory, 20 percent are failures and operative mortality is 4 percent. Only one of the failures has a true postphlebitic syndrome. Both operative fatalities occurred because of poor patient selection in that both patients had chronic congestive heart failure and both had suffered unrecognized massive pulmonary embolism prior to surgery.

Success of iliofemoral venous thrombectomy is correlated with both the preoperative duration of symptoms and the achievement of good bidirectional flow at operation. When results are tabulated according to the duration of symptoms, it appears that 80 percent of those patients with symptoms for less than five days had a good result as opposed to only 55 percent of those with a longer history. Among the 31 patients with good bidirectional flow at operation, 19 had an excellent clinical outcome with eight satisfactory results and two failures. Conversely, among the 14 patients with poor restoration of flow at operation there were only two excellent results, five satisfactory and seven failures.



Fig. 1

It is significant that it requires from two to five years for the postphlebitic syndrome to develop.[1,6,11] Considered in this light, the results among the 14 patients followed for more than two years are encouraging. One of the two failures had a poor surgical result initially and has developed the only true postphlebitic syndrome. The other failure had an excellent result for four months, suffered recurrent thrombosis which was managed conservatively and now has edema requiring continuous elastic support. Thirteen of the patients in this group were previously reported[18] and now, after an additional 27 months of follow-up, two excellent results have become satisfactory, one satisfactory result has become excellent and one failure has become satisfactory. No new failures have appeared.

TABLE IV

RESULTS OF ILIOFEMORAL VENOUS THROMBECTOMY

Operative Result:	Good 31 (69%)		Poor 14 (31%)	
	Excellent	Satisfactory	Failure	Death
Over-all Results	21 (47%)	13 (29%)	9 (20%)	2 (4%)
Results (excluding 2 patients with venous gangrene on admission and 1 patient with surgical ligation of common iliac vein)	21 (50%)	13 (31%)	6 (14%)	2 (5%)
Results in Cases with duration of symptoms < 5 days	18 (50%)	11 (30%)	5 (14%)	2 (6%)
Results in Cases with duration of symptoms > 5 days	3 (33%)	2 (22%)	4 (45%)	...
Results in patients followed > 2 years	5 (36%)	7 (50%)	2 (14%)	...

Average Duration of Follow-up: 21 Months.

Postoperative complications are presented in Table V. Delayed wound healing was usually caused by prolonged serous and lymphatic drainage. The high incidence of wound complications is undoubtedly related to both operative damage to lymphatics and the postoperative use of anticoagulants. The postoperative pulmonary emboli were both late, and the operative fatalities were related to poor patient selection.

TABLE V

POSTOPERATIVE COMPLICATIONS OF ILIOFEMORAL VENOUS THROMBECTOMY

Delayed Wound Healing	9 (20%)
None	29 (65%)
Wound Hematoma	2 (4%)
Wound Infection	4 (9%)
Pulmonary Embolism	2 (4%)
Operative Death	2 (4%)

Summary and Conclusions

Iliofemoral venous thrombectomy has three aims: relief of early morbidity, prevention of pulmonary embolism, and prophylaxis for the postphlebitic syndrome. On subjective clinical grounds the first goal has been achieved. The present series is too small to evaluate success in the prevention of pulmonary embolism, but it appears that surgery is at least as successful as conservative management in this regard despite legitimate, but groundless, fears. The achievement of clinical improvement in 76 percent of the entire group of patients is encouraging. And finally, the fact that 86 percent of these patients followed for more than two years have a good result justifies the cautious hope that the incidence of chronic deep venous insufficiency may have been materially reduced.

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Hospital Costs

Total hospital cost per patient day amounted to $55.82 in the hospital reporting year ending September 30, 1967, or a 14.3 per cent increase ($6.99) over the previous year, according to figures released in Hospital Indicators, a monthly report which appears in HOSPITALS, Journal of the American Hospital Association.

In March of this year the American Hospital Association prepared testimony for the House Ways and Means Committee on the Social Security Amendments bill affecting Medicare, and at that time AHA made a projection of hospital costs through September 30, 1967, which placed hospital costs per patient day at $57.93, an increase of 18.6 per cent over the previous reporting year.

After all the figures were collected for the 1967 reporting year the increase amounted to 14.3 per cent instead of the 18.6 per cent. "There are strong indications that the increase in hospital costs for this next year will be close to 15 per cent because of the continuing wave of salary increments for hospital personnel and the general increases in the cost of living."

Reticulum Cell Sarcoma of the Vagina

RANDOLPH HARRISON HOGE, M.D.
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Reticulum cell sarcoma of the vagina is a rare disease and is usually fatal. This case was treated with irradiation and chemotherapy.

PRIMARY RETICULUM CELL SARCOMA of the vagina is rare. We know of no case at the Medical College of Virginia other than the one herewith reported.

In 1961 Stringer and Penfield[1] in reporting a case of primary lymphocytic lymphosarcoma stated that a review of the literature to that date uncovered no other instance of primary lymphoma of the vagina, presumably including the reticulum cell variety.

At the time of our review of the American Literature of recent years the only case of primary reticulum cell sarcoma of the vagina found is that reported in 1958 by Weseley and Berrigan[2] in which a reticulum cell sarcoma occurred in the vagina in close proximity to the urethra in a 35 year-old Puerto Rican woman. Treatment consisted of local excision and postoperative external irradiation. Evidence of recurrence was present twelve weeks later.

In 1952 Ciulla[3] reported three cases. He advocated wide excision of the affected vaginal wall, followed by x-rays and the local application of radium. Two of the patients died following progression of the disease and one, discharged improved, was not followed.

According to an abstract in Excerpta Medica, Spampinato[4] in 1953, reported three cases of primary reticulum cell sarcoma of the vagina but details other than the diagnoses are not given in the abstract. Without at this time having access to the original article, it is not known if these three cases are the same as those reported by Ciulla.

In 1960, according to an abstract, Zapolla[5] reported a reticulum cell sarcoma of the vagina in a 48 year-old woman. Only pathological details are given.

In 1961 Van der Velden[6] reported the removal of a reticulum cell sarcoma from the vaginal wall of a 38 year-old woman; it had invaded the rectum. Six weeks after surgical treatment there was recurrence in the perineum and in the right side of the pelvis. Cobalt therapy led to rapid regression, but generalized reticulosarcomatosis developed. The patient died eleven months after the operation. At postmortem examination no tumor was found in the pelvis but there were distant metastases in many organs.

Case Report

Mrs. M. B., MCV #5-04-66-15, a 45 year-old, white, diabetic, obese housewife, mother of two children, was referred on June 13, 1963, following a biopsy of the vagina done in another city several days before.

In 1945 she had a bilateral tubal ligation. In 1953 she had a total abdominal hysterectomy, left salpingo-oophorectomy and appendectomy for leiomyomata uteri and endometrial polyps; there was no evidence of malignancy. In 1960 she had a cholecystectomy for chronic cholecystitis and cholelithiasis.

For several weeks prior to the present admission she had pain in the small of the

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back and in the left side of the abdomen. During this time she also had a vaginal discharge, but there had been no bleeding since the hysterectomy 10 years before. For sometime she had had "the feeling that everything was going to fall out" of her. For about a year she had had diarrhea and constipation.

The general physical examination was unremarkable. Pelvic examination was as follows:

The vaginal outlet was large. The entire mucosa of the anterior vaginal wall appeared abnormal. (Fig. 1) It was thickened and



Fig. 1.

had a nubby surface especially near the introitus. The mucosa in the vault was indurated and discolored. The mucosa was intact everywhere except for three biopsy wounds. The posterior wall seemed to be free of disease. The uterus was absent, and there was no sign of disease in the adnexa or parametria.

Her temperature, pulse, respiration and blood pressure were normal. The hemoglobin, the blood smear, the white count and the differential count were normal. The urine showed sugar and the fasting blood sugar was 191 mg %. On chest x-ray the lungs were clear.

Biopsies were performed on June 14. The pathologist reported that there was no ulceration of the mucosa and its basal cell layer was intact. Underlying the mucosa there was a diffuse compact infiltration of small, regular, oval cells with prominent nucleoli sequestrated in a dense fibrous stroma. Mitotic figures were rare and pleomorphism minimal. Lymphosarcoma (reticulum cell type) of the vagina was the pathological diagnosis. (Fig. 2)

An intravenous pyelogram on June 17 was essentially unremarkable, and cystoscopic examination on June 20 showed smooth bladder walls with one raised hemorrhagic area. Biopsy showed acute and chronic cystitis. On the same day bilateral lymphangiography was done. The lymphatic system showed up nicely all the way from the pelvis to the mediastinum, and there was no evidence of involvement with tumor.

A decision was made to treat the patient with x-ray and to limit the treatment to the pelvic and lower para-aortic areas. Accordingly, over an elapsed period of 24 days, she received 19 treatments totalling a 3800 roentgen tumor dose delivered by a two million volt maxitron through an anterior and a posterior portal.

She was discharged from the hospital on July 12th definitely improved and was to return for a postoperative examination. This she failed to do. She was examined by her local doctor on January 27, 1964, seven months after her treatment; he reported a "minimal roughening and induration in the suburethral area" with no obvious local changes on inspection.

On June 15, 1965, two years after treatment, the patient was referred for a follow-up examination. She had noted some bloody spots on her underclothes from time to time and complained of some bladder trouble. Pelvic examination then showed the vagina to be smooth and soft except just within the introitus on the anterior vaginal wall where there was some thickening and nubbiness somewhat similar to the condition noted prior to treatment but it was less marked. The rest of the pelvic examination was negative.

She was re-admitted to the hospital on July 12, 1965. The vagina was biopsied and the pathologist found only mild vaginitis.

There was no evidence of sarcoma. An intravenous pyelogram was negative and a cystoscopic examination showed some granulation tissue at the bladder neck and some cystitis. No sarcoma was noted. The admission hemoglobin was 13.2 grams and the white blood count 5,300. She was discharged from the hospital on July 21, 1965, apparently free of the disease.

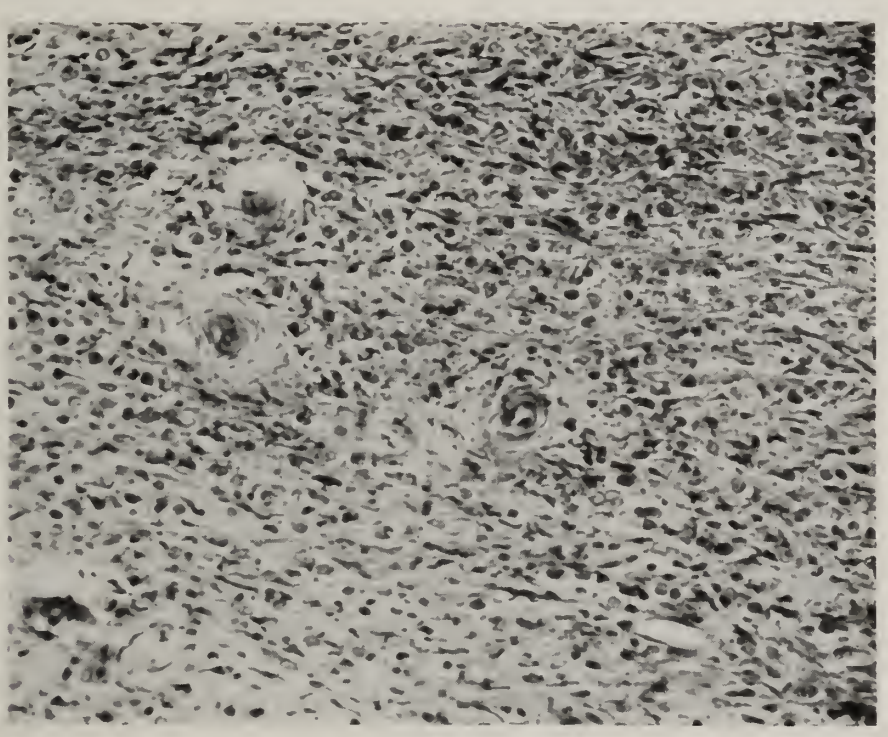


Fig. 2.

On April 1, 1966, this patient was readmitted to the Medical College Hospital with a lesion of the right maxilla and gingiva, a hard right supraclavicular mass, and a mass in the inferior medial quadrant of the right breast. Pelvic examination was negative.

Biopsies of the mass of the breast and the oral mass revealed reticulum cell lymphosarcoma. The patient then received irradiation to the involved areas and chemotherapy with cyclophosphamide. She was discharged from the hospital on May 31, 1966.

She returned to the hospital on August 2, 1966, with tumors involving the full extent of the vagina and a residual lesion in the right breast. X-rays of the chest and bones and cystoscopic examination were negative. She was again given chemotherapy and discharged from the hospital.

She died elsewhere on September 26, 1966, three years and three months after the original diagnosis was made. A death certificate recorded "metastatic sarcoma of the vagina" as the cause of death. Autopsy was not performed.

Summary and Conclusion

Reticulum cell sarcoma of the vagina is a rare and generally fatal disease. Treatment in most cases has been excision and/or irradiation. Chemotherapy also was used in our case. Young and Finkle[7] have found Vincristine of value in advanced cases of reticulum cell sarcoma which previously had been resistent to other forms of treatment. Their cases did not include reticulum cell sarcoma of the vagina. Cases of reticulum cell sarcoma of the female genitalia other than in the vagina are described in the literature[8,9,10] but are not a part of this presentation.

A case is herewith reported of reticulum

cell sarcoma apparently primary in the vagina. The patient initially responded well to external irradiation but later there were wide spread metastases, local recurrence, and a fatal outcome.

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Dr. George Prout aided in the lymphangiography, Dr. Elmer Bear biopsied the maxilla, and Dr. Susan Mellette prescribed the chemotherapy.

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Foreign Body Embolus to Middle Cerebral Artery

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This is an interesting and unusual case of foreign body embolus with symptoms appearing 12 years after the original injury.

FOREIGN BODY arterial embolization has been reported frequently, with the lower extremity being the most usual site of involvement.[1,2] Harkin in his review of 134 cases,[3] reports none with embolization to the brain; and indeed, until 1960 only three such cases were reported in the literature.[4] In these patients there was complete occlusion of the middle cerebral artery and the onset of symptoms followed quite rapidly upon the acute injury. Radiological documentation was obtained in only one case. This patient would therefore appear to be unique, a angiographic delineation of an incomplete occlusion from a foreign body embolus in this location has not previously been reported.

Case Report

The patient is a 30 year old, right-handed, male, admitted to the Neurology Service of the Naval Hospital, Portsmouth, on January 25, 1966, for evaluation of persistent headaches. He stated that he had been previously well until approximately seven months prior to admission when he experienced the abrupt onset of right sided, retro-orbital, throbbing headaches, occurring at a frequency of approximately twice a week, and lasting for several hours; they were unaffected by position change, coughing or straining.

The most significant feature of the past medical history was that in October, 1954, (while making a rapid egress from a female companion's boudoir) he received a shotgun injury to the left arm and chest, and was hospitalized with a hemopneumothorax. Chest x-rays at that time revealed, in addition to the hemopneumothorax, multiple metallic pellets over the left lung field, mediastinum and shoulder. The patient recovered uneventfully.

Admission physical examination revealed a well nourished male with multiple punctate scars over the left shoulder, posteriorly. Except for the findings on neurological examination, the remainder of the physical and laboratory data were normal. Neurological examination revealed a mild weakness and reflex hyperactivity confined to the left upper extremity, associated with what was felt to be a mild organic mental syndrome. An electroencephalogram was abnormal and revealed the presence of small amounts of right temporal slowing, exaggerated by hyperventilation.

Dr. Sandok and Spiegel were formerly with the Neurology and Radiology Services of the Naval Hospital, Portsmouth, at which time this article was written.

The opinions or assertions contained herein are those of the authors and are not to be construed as official or reflecting the views of the Navy Medical Department or Naval Service at large.

Plain skull films (Fig. 1) revealed no defect in the vault. A single round metallic



Fig. 1. Lateral film of the skull, together with A-P projection (not shown), the foreign body localized in middle cranial fossa, lateral temporal region, just at the point of origin of the sylvian vessel.

foreign body was present in the right temporal area. PA (Fig. 2) and lateral films of the chest on this admission revealed multiple



Fig. 2. PA chest, present admission.

shotgun pellets in the soft tissues of the back, middle and superior mediastinum, and the left lung field. Previous chest films were not available. From present films and the available history, the radiologist was able to suggest the apparent source and approximate location of the embolus. A right carotid arteriogram (Figs. 3 & 4) revealed that the pellet lay in the middle cerebral artery just proximal to its trifurcation. Contrast material appeared in good concentration distal to the pellet outlining normal arterial trunks without evidence of distal occlusion.

Surgical removal or other therapy was not deemed advisable at this time, and the patient was discharged from hospital care.



Fig. 3. Right carotid arteriogram, half axial view. The shot is clearly visualized with excellent opacification of the sylvian vessels distally.

Discussion

The exact site of origin of the embolus is uncertain; however, several alternative pathways exist. Direct entry into the carotid or middle cerebral artery can be excluded in this case since there were no penetrating wounds of the head or neck on the side of the intracranial pellet. Entry into the heart or ascending aorta is the most frequent source of foreign body emboli.[1,3] This latter even may occur without significant morbidity.[5] It is, of course, conceivable that one

of the fragments entered the heart by perforating a major pulmonary vein; however, this has not yet been documented as a mode of entry. Intravascular entry need not occur acutely, but may appear as a consequence of gradual erosion of a foreign body through the vessel wall; this is more com-



Fig. 4. Right carotid arteriogram, lateral view.

monly seen with sharp or elongate fragments.[6] Yet another possibility is that embolization can be delayed by entrapment of the foreign body in the chorda tendinae or ventricular lumen.[7]

The rather abrupt onset of the patient's symptoms 12 years after the original injury, suggest that delayed embolization had occurred.

Summary

A case of delayed foreign body embolization to the middle cerebral artery is presented. The embolization resulted in incomplete occlusion of the middle cerebral artery just proximal to its trifurcation.

Only three previous reports deal with foreign body embolization to the internal carotid system. This would appear to be the first case in which there was angiographic demonstration of an incomplete occlusion.

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Let's Reminisce!

THE TRANSPLANTATION OF BONE—The greatest discovery in surgery, thus far in the year 1881, is that of Dr. William MacEwen. He has successfully transplanted bone—fragments of wedges of bone taken from patients for curved tibiae—into the arm of a child whose limb was useless by reason of extensive necrosis; two-thirds of the humerus had been destroyed and no repair of bone had taken place. A good new humerus was the result, less than an inch shorter than its fellow.—Proc. Med. Soc., Co. Kings, August, 1881. (reprinted in the Virginia Medical Monthly, August 1881)

Prescribing a Camp for the Handicapped Child

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There are approximately one million handicapped children of camping age in the United States. Many can benefit by being placed in the appropriate camp.

SUMMER CAMPING is widely acknowledged to be beneficial to children, yet relatively little is known about camping experiences available to children with handicaps. More specifically, few parents and their medical advisors make use of the hundreds of camps throughout the United States which are oriented toward the child with handicapping conditions. For example, it is estimated that there are twelve thousand children in the United States between the camping ages of 7-15 years with diabetes mellitus. Only 17% of these in 1966 took advantage of the forty-five diabetic summer camps scattered throughout the country. There are many reasons for this, including financial status, fears concerning the child leaving home, and vacation conflicts; however, it is strongly felt by many that lack of communication to the parents regarding camping benefits to a child with a handicap may be the primary reason for the relatively small percentage of participation.

The goals of a camp for handicapped children can be classified into three groups —recreation, education and research.

Perhaps the single most beneficial event is the separation of the handicapped child from home to a recreational environment. Most handicapped children's camps provide a safe, well supervised, structured recreational schedule which is nearly impossible to achieve at home. At Camp Kiwanis, for handicapped children on the Blue Ridge Parkway in Virginia, fishing, swimming, short hikes and overnight camping in Virginia's beautiful Blue Ridge Mountains were carried out for children with handicaps ranging from cerebral palsy to rheumatic fever. These alone were the camp's main objectives—a new adventuresome experience. It has been said that the personality is derived in large measure from one's life experiences. Imagine the "boost" a handicapped child's personality receives from adventuring the outdoors. Most camp directors will tell you that the single achievement mentioned most often in letters from the family of campers following this experience is the boosting of the child's self image.

This objective of camping is something which any child may gain; the handicapped child may need it more often due to a previously sheltered home environment. Singular to handicapped children's camps, however, is the opportunity to have specialized personnel and facilities for education with the child's specific problem. Rehabilitation in the areas of speech and hearing, orthopedic deformities and emotionally disturbed children has made great strides in a camp environment. The Diabetic Children's Camp is one of the most notable examples. The juvenile diabetic can make significant gains

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caring for himself. Training regarding Insulin shots, urine testing, and sterile technique if often neglected at home and the parents bear the brunt of the child's care. During the camp session emphasis is placed on the child assuming responsibility for his own self care under medical supervision. This same concept of giving handicapped children more responsibility can apply to any camp setting.

The availability for staff learning and research is obvious where a group of children with common medical problems. Much can be learned about the individual child, and the referring facility may be furnished with information in regarding the individual camper, his peer relationships, and of his strengths and weaknesses regarding his ability to overcome his handicap. Informative research papers have been published from the observations made at summer camps for handicapped children.

Work has shown that children with a variety of handicaps can adapt well to usual camping circumstances. It has also been demonstrated that the training of camp counselors and other staff members is relatively easy, yet permits these children to enter into a wider range of activities with greater ease and fewer traditional limitations.

Not mentioned in the previously discussed goals is the well deserved rest the parents have earned for the few weeks the child is away from home. This gives them a chance to relax and re-examine themselves in relation to the child. The separation in itself provides the opportunity for a more objective appraisal of the child-parent relationship and the child's unique characteristics.

Having reviewed the goals of a handicapped children's camp some examination next may be given to the acceptance of a handicapped child into the non-specialized camp. Put another way, can the child with a definite handicap be integrated with the recreational camp with a predominance of children without handicaps? Another question that might be asked, would it be better to send a child with a limited handicap (seizures, diabetes, "withered arm") to a specialized camp or to a non-specialized camp?

The authors conducted a survey in Virginia of the 212 licensed children's summer camps. We received replies from 165 camps. (77%) The primary objective was to accumulate some data on the following questions: (1) How many non-handicapped children's camps accept children with handicaps? (2) What degree of limitation and type of handicap is most often accepted by the non-specialized camps? (3) What are the primary reasons for not accepting a handicapped child to a non-specialized camp?

Our findings revealed that over two-thirds of the non-specialized camps in Virginia accept children with handicaps; however, in reviewing the data more closely, one half of these camps accepted only children with minor handicaps such as allergy, asthma, hearing loss, hay fever, etc. Below are the percentages of various types of handicaps accepted by most non-specializing camps:

Arthritis	29%	Allergy	53%
Visual defects	39%	Speech & hearing	55%
Emotionally disturbed	28%	Cerebral palsy	18%
Pulmonary disease	9%	Heart defects	25%
Epilepsy	29%	Hemophilia	10%
Mentally retarded	33%	Orthopedic handicaps	28%

There are 39 camp directors who gave reasons for not accepting handicapped children. Seven of them felt it would be a source of embarrassment to the other children. All of them felt it demanded too much of the staff and facilities available. Fifteen thought that some parents of the children without handicaps would not wish to place their child in an environment with handicapped children. Twenty reported

that camp surroundings may be too dangerous for a handicapped child.

Of the camps accepting handicapped children we were interested to examine the camp director's opinions on the relationship between degree of limitation of the child's defect as compared to the benefit gained from the camp experience. Put another way, how limited may the handicapped child be before he loses out on the benefits of a camping experience? Fifteen percent felt that the handicapped camper should be able to participate in most activities with brief rests with an omittance of an occasional strenuous activity. Only 24% thought that sedentary activities alone would be sufficient.

In examining the results of this study, there are some points which may be discussed. If non-handicapped children's camps in Virginia are a representation of those throughout the country, it is refreshing to find that a significant number of them do accept campers with various handicaps. It was felt by most respondents that the majority of children would not benefit from camp without being able to participate in most activities, although a range of limitations was offered.

Many physicians now involved with a child having a limited handicap are beginning to lean towards the concept of gradually working these children into a "normal" environment rather than isolating them. For example, a twelve year old boy may have had diabetes mellitus for half of his life. Over the past two years he had attended a diabetic camp with good cooperation by the child, parent, and family physician, and achieved the ability to handle his condition well. He wants to go to camp; he has reached a plateau in the learning received from the diabetic camp, however. The State Office of the American Camping Association headquarters is contacted and a recreational camp is chosen which is acceptable to the child.[1] The family physician and previous diabetic camp director contact the recreational camp director with letters of recommendation. With the working together of these people—parent, family physician, and camp directors, a boy such as this may be progressed to a new of achievement as he matures with training and teaching. This same concept may apply to most handicaps varying from seizures to orthopedic deformities.

In prescribing a camp for a child with a specific problem the following things may be considered: (1) The objectives of the child's camp experience should be established. (2) The family may need advice and counsel from outside personnel. (3) Financial assistance may be needed from various organizations. (4) A follow-up evaluation and reinforcement should be conducted.

First we must establish the goals—what does the child need? A girl with an isolated speech problem might best be handled at a specialized camp for a few years. The speech therapist and family physician could be most helpful in laying out the objectives for the child on a long and short term basis. Specialized assistance may be received from the local health department personnel, local physician, social workers, or a number of state and national headquarters oriented toward camping for a handicapped child.[2]

Total or partial financial assistance may be obtained by a sponsorship from local civic organizations. A civic directory may be obtained from the local Chamber of Commerce and letters regarding the child sent to the various organizations. Also federal funds may be obtained from one of three sources—The United Givers Fund, The Mayor's Council on Youth Opportunity, Community Action Program.

Not the least of importance, a follow-up of the handicapped child should be carried out by the referring personnel. Evaluation of the camp experience and reinforcement can be very beneficial. The gains made at camp may be built on and further developed by the referring facilities.[2]

From the recent (1964) population estimate by the Bureau of Census it is estimated that there are thirty-four million children in the United States today of camping age (between 7 to 15 years old). Approximately 3% or one million children of camping age are handicapped children. It is hopeful that the trend towards enrolling these children in specializing and non-specializing camps will continue.

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Motor Vehicle Fatalities at High Level

Continued growth in the use of motor vehicles was again demonstrated last year when new peaks were reached in the number of motor vehicles registered, drivers licensed, and vehicle miles traveled. During the last five years the number of drivers increased from 90.5 to 102 million.

The largest proportionate increases in the numbers driving occurred at ages under 20 and among those 65 years and older. There were approximately 10 million drivers at ages under 20 in 1966, or about 60 percent more than five years ago; the number of elderly drivers rose by a third to an estimated 8 million.

The rise in the volume of motor vehicle traffic has been accompanied by a steadily mounting number of fatalities. The death toll has climbed to a new high in each of the last five years, from 40,800 in 1962 to an estimated 53,000 in 1966. The experience in 1967 to date suggests that the trend may have been at least temporarily halted even though the motor vehicle accident death rate remains at about 27 per 100,000 population.

Among males at ages 15-19 the motor vehicle fatality rate rose by almost a fourth in the period under review, while at ages 25-64 it increased by about a fifth. At ages 65 and over the corresponding increase in the accidental death rate was only 11 percent and for young men in the age range 20-24 it was 13 percent; the motor vehicle fatality rate among the younger men is far higher than at any other period of life, about 80 per 100,000 population. The next highest fatality rates have been recorded among men 75 and over and boys 15-19.

The increases in motor vehicle fatality rates were greater among females than among males. By age they ranged from 16 to 41 percent, except that the accidental death rate for girls at ages under 5 showed little change. Fatality rates remain, nevertheless, substantially higher for males than for females.

A greater toll among drivers and passengers accounts for practically all of the higher motor vehicle accident mortality among both adolescents and adults.

The hazard of fatal injuries in motor vehicle accidents tends to increase with the size of cities. Data compiled by the National Safety Council show that in four of the past five years the highest fatality rate per 100,000 motor vehicles registered occurred in cities of over one million population. Last year these large cities and those with populations of 500,000 to 750,000 recorded identical rates of 32 per 100,000 motor vehicles registered, whereas the group of small towns with populations of 10,000 to 25,000 had the most favorable record with a rate of 19 per 100,000. Despite growing urbanization, more than two thirds of the fatalities due to motor vehicle accidents occur in rural places. (Statistical Bulletin, Metropolitan Life Insurance Company)

University of Virginia's "Firsts" in the History of Medical Education

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THE UNIVERSITY OF VIRGINIA School of Medicine in Charlottesville has played a prominent and unique role in the history of American medical education. The following contains a brief review of some of the "firsts" which have now become commonplace in medical schools in this country.

The University was chartered by the General Assembly in 1819 under the sponsorship of Thomas Jefferson and opened for instruction in 1825. Among the eight "Schools" founded at that time was the School of Anatomy and Medicine, established in 1824. The first graduates received their diplomas in July, 1828.

A Full-Time Teaching Staff

Although it has frequently been stated that the first full-time faculty of five professors was employed at the University of Michigan during the 1850's, the appointment of Dr. Robley Dunglison at an annual salary of $1,500, tuition fees ranging from $25 to $50 per student, a house and a garden, indicates that this honor should go to the University. Dr. Dunglison was truly "the first full-time professor of medicine in an American University", and "A novel feature of Dunglison's contract with the university, unheard of at the time in America, was the restriction of his practice outside the university to consultation, a means of assuring maximum attention to the needs of the medical students." [1] Dunglison did not seem to have objected to these restrictions. As reported by two Charlottesville historians, "From 1825 until 1833 Dr. Dunglison was a full time professor of medicine, thus restricted to a very limited practice, but as he emphasized, . . . 'a restriction which I greatly preferred.' " [2] In 1833 Dunglison accepted an appointment at the University of Maryland, and three years later he moved on to Philadelphia as Professor of the Institutes of Medicine and Medical Jurisprudence at Jefferson Medical College.

During Dunglison's tenure in Charlottesville, the teaching staff was expanded by two professorships. In 1827 Dr. John Patton Emmet became head of a new School of Chemistry and Materia Medica. Emmet had come to the University in 1825 as Professor of Natural History. The terms of his employment were identical with those offered to Dunglison, *i.e.*, a salary of $1,500 a year, in addition to tuition fees from students, a house and a garden. Moreover, the Visitors, in 1827, assigned the Departments of Practical Anatomy and Surgery to a demonstrator, Dr. Thomas Johnson. Dr. Johnson's salary was set at $500 a year, the use of a house, and a $15-fee per student. In 1829 Johnson was made a member of the faculty, and in 1832 he was advanced to a regular professorship.

The stringent prohibitions against income from sources other than from the salary, student and consultant's fees were maintained for many years. The *Catalogue of the University of Virginia* for the session of 1848-49, for example, stated: "The enactments of the Visiters [sic] of the University prescribe that no Professor shall engage in pursuits of emolument unconnected with the service of the University, or shall receive from the members of his class any compensation in addition to that provided for by the laws." [3] These standards must be viewed as unusual at a time when many medical

teachers at other schools lacked fixed annual salaries and received most of their income from fees of students and private patients and from apprentices who, in some instances, were also students at their respective medical schools.

Length of the Academic Session and Graded Curriculum

Another noteworthy difference between the system of medical education at Virginia and other medical schools of the early 19th century pertained to the length of the academic session. In his celebrated book *The Medical Student,* published in 1837, Dunglison wrote as follows: "The session of the chief Medical Schools generally commences about the 1st of November, and terminates on the last day of February. Recently, the University of Pennsylvania has extended the session to the end of March. The anatomical rooms are sometimes opened earlier than the commencement of the session." [4] He then listed the dates at which "the Lectures commence" in some twenty-three schools. On the other hand, speaking of the University of Virginia, Dunglison said: "Unlike the mass of medical schools, the session of the medical department of the University of Virginia is of the same length as the academic session, ten months."

The length of the session was later on reduced to nine months and the students had to attend two lectures a day. That method, according to the *Catalogue* for 1848-49, gave the students ample time for private reading and anatomical dissections. It was also pointed out that this system contrasted sharply with that in use at "nearly all" other medical schools of the country whose terms of instruction were generally limited to periods of four months. Due to the large amount of instruction which had to be compressed into such a short period, the *Catalogue* stated that the students were forced to listen all day long to " . . . lectures delivered in rapid succession, and treating of diverse topics".

The system, moreover, was said to preclude a graded curriculum, because "the lectures on the different branches of medicine are carried on simultaneously." At the University of Virginia, on the other hand, the "fundamental branches of Medical Science" were stressed before the students' "attention is directed to their practical application", and " . . . the fundamental branches of Medical Science are precisely those which demand for their illustration the apparatus only to be found within the walls of colleges." According to these concepts, therefore, the medical student at the University of Virginia had to take the courses in anatomy, physiology, chemistry and materia medica " . . . before he is required to apply these branches in the study of the Principles and the Practice of Medicine."

The fact that most of the instruction was given in the form of didactic lectures—except for practical demonstrations in the out-patient teaching clinic and "dissection of fresh subjects with which the School is abundantly supplied"—should not detract from the basic concepts underlying the method of instruction at the University. Today the academic terms of most medical schools parallel those of the other faculties of their respective schools. Such practices were not known at a time when, according to the University's *Catalogue* for 1848-49, "Nearly all the medical schools of this country . . . have only a nominal connection with the colleges from which they borrow their names and chartered privileges." The graded curriculum has also become a part of the present-day medical school curriculum, although there is some disagreement among educators concerning the amount of free time which the medical student should have for reading and discussion. The idea that instruction in the pre-clinical disciplines should precede the clinical terms is also generally accepted.

A Diploma in the English Language

One of the lesser known innovations at the University was the issuance of a diploma

in the English language. Dr. Dunglison, in *The Medical Student,* commented as follows: "As regards the diploma, the University of Virginia has commenced the innovation of writing it in English, so that it may be intelligible to every one."

It is to be observed that this break with tradition came at a time when most universities issued their diplomas in Latin.

The question of the knowledge of Latin as a prerequisite for graduation from the University was a subject of much discussion in Charlottesville during the initial stage of the University. Originally a rule existed which denied a diploma to any student "who had not passed such an examination in the Latin language, as proved him able to read the highest classics in that language with ease, thorough understanding and just quantity." [5] Dunglison objected to the rule, among other things, on the ground that the requirement would "retard student enrollment and, consequently, the emolument of the Professor of Medicine." As a result of this protest the Board of Visitors, with Thomas Jefferson voting against the motion, abolished the requirement in favor of one to the effect that any candidate for graduation give "satisfactory proof of his ability to write the English language correctly." This decision (of 1826) should not be interpreted to mean that Latin was not held in high esteem at Virginia. But it seems that the newly founded American educational institutions of the early 19th century would not stress classical studies to the degree to which they were cultivated at most of the European universities in which the medical student was still instructed in Latin.

Writing in *The Medical Student* Dr. Dunglison said that " . . . a certain knowledge of that tongue [Latin], as well as of Greek, ought still to form part of the education of every gentleman." It was also necessary " . . . as to be able to comprehend, after a little instruction, the various directions that may be given for the preparation of extemporaneous and officinal formulae."

It is clear that the issuance of a diploma in the English language at that time represented a fundamental change in university traditions. Dunglison was very much in favor of the new practice pointing out that a lesser degree in a foreign language could easily be represented as a doctor's degree to someone who did not have the knowledge of that language. In short, frauds could be prevented, especially in states in which graduates of a medical school were permitted to practice without further examination. Dunglison also deplored the verbose style of many diplomas in vogue then. He concluded that, " . . . no matter what may be the fancy, we think it more judicious, that the diploma should be written in English."

Dunglison pointed out that "In the establishment of the University of Virginia, the venerable customs that had prevailed for ages were retained only when esteemed worthy of retention. Precedent was wholly disregarded." These sentiments may sound strange in a day when the University and its founders are often regarded as bastions of conservatism. Actually Thomas Jefferson and other Virginia contemporaries were forward looking innovators who had no qualms about discarding "venerable customs".

Teaching the History of Medicine

Medical historians will take pride in the fact that the first course in the history of medicine to be held at an American medical school was given at the University of Virginia. "The history of the progress and theories of medicine" was one of the subjects which Dr. Dunglison undertook to teach when he agreed to accept the first professorship in medicine at the University. The reason for this innovation was Thomas Jefferson's desire to instill in the student not merely a knowledge of the fundamental branches of medical science, but also " . . . something of the earlier progress of the science and the art". This was in line with

Jefferson's broad concepts of education. Dunglison's lectures at Virginia were published posthumously in 1872 by his son Richard J. Dunglison, M.D.[6] They covered a wide spectrum of medical history, including such unusual topics as "Medicine of the Hindoos", "Medicine of the Scythians", and "Medicine of the Celts".

Few American medical students today will have an opportunity to obtain such a fine background in the history of their profession. The teaching of that subject had been discontinued at the time when Dr. Richard Dunglison compiled and edited his late father's lecture notes in 1872. In the preface he wrote: "The students of those times were, therefore, in this particular, a step in advance of the condition of their successors of the present day, who are left to gather their information on the previous state of medicine in whatever manner they may find it practicable or convenient to do so, after graduation."

Jefferson and Dunglison may have been trail blazers here too, however, because the teaching of medical history is receiving increased attention in recent years and there is a good possibility that this subject will be incorporated into the curricula of American medical schools.

Jefferson passed away on July 4, 1826, only a little over one year after the doors of his beloved "academic village" had opened. The innovations in medical education which are the subject of this paper have given the University a distinguished place in the annuals of the history of medicine of the United States.

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Library, School of Medicine
University of Virginia
Charlottesville, Virginia

Let's Reminisce!

FOR SALE—Many of our readers will be glad to know that some of the articles to be named or books or journals are purchasable. They are offered for sale by one of our most useful contributors or subscribers. Any communication addressed to the Editor of the *Medical Monthly* on the subject, especially those relative to prices, will be at once communicated to the gentleman who has the articles for sale. He offers a first-class obstetric manakin, made of the finest buckskin, open to button over the abdomen. It is not soiled in the least. The representative foetus is slightly torn at the neck; but it has a good cord and a good placental substitute. The entire manakin is enclosed in a nice wooden case. The manakin representation of the woman has straps on her back "to keep her from getting out of bed" during the second stage of labor. It would be well for a medical college or a tutor, instructor or professor of obstetrics to remember this. (Virginia Medical Monthly, July, 1882)

Clinicopathological Conference . . . .

Acute and Chronic Abdominal Pain in an Elderly Female

Prepared and Edited by

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Clinical History

A 72 year old disoriented Negro female was admitted to the Medical College of Virginia Hospitals on 3-5-65. Her family said that she had been complaining of mid-abdominal and back pain for over a year. Her appetite had been poor and she had lost weight. There had been no constipation, fatty food intolerance or ulcer symptoms.

Eight days prior to admission she had increasing abdominal pain and stopped eating altogether. She developed diarrhea and vomiting and, two days before admission, began to have bloody diarrhea which persisted.

Past medical history revealed an appendectomy many years before and she had "heart trouble" for which she took a pink pill every day.

Review of systems showed dyspnea on exertion and at rest. She had occasional ankle edema.

Physical examination: BP 160/90, P. 140, R. 30, T. 100 (R). The patient was an acutely ill, elderly woman moving restlessly in the bed. She was disoriented as to time and place, but complained of abdominal pain. Her respirations were labored and rapid. The neck was supple. She was edentulous and her mucous membranes appeared dehydrated. The lungs were clear to percussion and auscultation. The cardiac apex was in the 5th left intercostal space just outside the midclavicular line. There was regular rhythm and no murmurs. The abdomen was slightly distended and there was generalized tenderness without guarding. No masses were detected and there was no rebound tenderness. Bowel sounds were hypoactive. Rectal examination was negative except for bloody stool in the ampulla.

Laboratory data: The urine was amber, slightly cloudy and acid with a specific gravity of 1.018. There was a trace of proteinuria, but sugar and acetone were negative. Microscopic examination showed 6-7 WBC/hpf and 4-5 RBC/hpf. Hemoglobin was 15.2 grams percent, WBC 42,000/mm^3 with 87 percent neutrophils, 11 percent lymphocytes and 2 percent monocytes. BUN was 59, blood sugar 115 mg. percent. Serum sodium 134 mEq/1, chloride 91, potassium 3.8 and CO2 combining power 23. Serum amylase was 204 Somogyi units.

An electrocardiogram was reported as showing supraventricular tachycardia.

A chest film showed a density in the right apex and along the right mediastinum which displaced the trachea to the left.

A flat film of the abdomen showed no free air although there was gas in the stomach and large bowel. Fluid levels were present in the small bowel.

An attempt at sigmoidscopy was unsuccessful because of poor preparation of the patient.

The patient was treated with intravenous fluids and antibiotics but her condition seemed to be deteriorating. A laparotomy was performed 10 hours after admission.

Clinical Discussion

Dr. Max Rittenbury: I feel somewhat like the 65 year old man who came to the new Medicare Clinic. As he walked in the front door he found several doors facing him. The first of these said "Appendicitis", the second "Heart Disease", and the next "Cancer", etc. He figured, "Well, I've got a stomach ache so I must have appendicitis", and he went through the door marked "Appendicitis" but came to two more doors. One of these was marked "Male" and the other "Female". Naturally, he went through the "Male" door and lo and behold, he came to two more doors. One was marked "Protestant" and the other was marked "Catholic". He was a Protestant so went through the "Protestant" door and faced another choice. This was "Taxpayer" vs "Nontaxpayer". He had a small house, he paid taxes, so he walked through the door marked "Taxpayer" and he saw "Married" and "Single" on the next two doors. He was getting a little tired of this, but figured it was part of the folly of the Great Society and since he was married, he walked through the door marked "Married" and finally came to two more doors. One said "Republican" and one said "Democrat"; a life-long Republican, he opened the door and stepped through and immediately fell nine floors to his death. So, I feel somewhat like this gentleman as we go through the differential diagnoses, although I hope the ending will not be quite that bad.

This was a 72 year old, disoriented, Negro female, who was admitted on March 5th. Her family said that she had been complaining of midabdominal pain for over a year. Her appetite had been poor and she had lost weight. There are several questions that should have been asked while taking the history. Where was the back pain? Was it between her shoulders or was it down around the sacrum? Was the pain cramping or constant? Was it sharp and was it caused by movement? The additional history given is that she had no constipation, fatty food intolerance or ulcer symptoms. I am not really sure what "ulcer symptoms" mean in this case but I am going to assume that it means epigastric burning or eructation. Now, eight days prior to admission she noted increasing abdominal pain, which I am assuming was like that she had had before. She stopped eating altogether. She developed diarrhea and vomiting and, two days before admission, began to have bloody diarrhea. Several other facts would be very helpful here. What came up when she vomited? What was the color, the content? What did it taste like? Also, how frequently did she vomit? Did she still try to take in food or water? How frequent was her diarrhea? Was it one, two liquid stools a day, or was it multiple liquid stools a day?

The past medical history revealed that she had had an appendectomy many years before and that she did have heart trouble for which she took the usual pink pill per day. Review of systems was essentially negative, apparently, except for dyspnea on exertion and at rest. She had some occasional ankle edema and I think this would fit with her age and mild congestive heart failure. The patient was an acutely ill, elderly woman, restless, disoriented but complaining of abdominal pain. Her disorientation, as in any senile or elderly patient, could certainly have been due to vascular disease. However, these patients will usually have retinal arteriosclerosis, previous complaints of headaches and other evidence of arteriosclerosis. Toxic delirium or toxic disorientation will frequently occur in the elderly patients in association with the acute illness and is probably the situation here.

She was edentulous and her mucous membranes appeared dehydrated. I think that the use of the mucous membranes as a clinical sign of dehydration is open to serious

questioning because people who are breathing rapidly or with their mouths open can frequently dry out their mucous membranes. The turgor of the skin and the general appearance of the patient can give you a better clinical sign of the state of dehydration than just the condition of the mucous membranes itself.

We come to the major system involved. The abdomen was slightly distended and there was generalized tenderness without guarding. No masses were detected and there was no rebound tenderness. Bowel sounds were hypoactive and the rectal examination was negative except for bloody stool in the ampulla. There is nothing in the history about the size of the patient. Is she a very thin, elderly female? If so, the fact that you found no masses would be of little significance, particularly in somebody who has tenderness, even without guarding.

Dr. John H. Moon: She weighed less than 100 pounds.

Dr. Rittenbury: Her generalized tenderness without guarding, considering her size and age, is compatible with acute peritonitis. Frequently elderly, somewhat debilitated people mask their pain and do not show the signs you would expect to see with generalized and diffuse acute peritonitis in the younger person. I will assume that the pelvic examination was negative. The rectal examination was negative except for bloody stool in the ampulla.

The relatively normal urine would tend to make me believe her vomiting was not excessive and that she was not severely dehydrated or starved. Microscopic examination of the urine did show 6-7 WBC/hpf and 4-5 RBC/hpf. The hemoglobin was 15.2 with the white count 42,000 and 97 percent neutrophils and is suggestive of a moderate degree of dehydration with an infection or acute inflammatory reaction. The elevation of the BUN at 59, the elevation of the hemoglobin, the blood in the intestine and also the moderate specific gravity of 1.018 should make you think that this person was only moderately dehydrated and had some impaired renal function. The mild electrolyte alterations are consistent with her history of vomiting and diarrhea. Serum amylase was 204 Somogyi units and I am not really sure of what that means. The upper limits of normal here is 150; however, it must go up much more before I attach any great importance to it. Renal impairment with poor excretion of amylase might account for the deviation.

An electrocardiogram was normal except for a supraventricular tachycardia. Attempt at sigmoidoscopy was unsuccessful because of poor preparation of the patient. When this is so and you feel it is important to proctoscope a patient who had bloody diarrhea and/or soft stools, there is a long suction apparatus that can be used at the time that you scope these patients and you can remove quite a bit of the contamination from the rectum. These are available and will be sent to the emergency room if you scream loud enough. The patient was treated with IV fluids and antibiotics but her condition seemed to be deteriorating. A surgeon was called in and a laparotomy was performed.

Could we see the x-rays?

Dr. M. Pinson Neal: On the chest film we see evidence of marked arteriosclerosis of the aorta, and a relatively normal appearing heart, considering her age. The bones are demineralized. A look at her dorsal spine reveals a scoliosis with some degree of rotation. The mass alluded to in the protocol represents to me a buckling of the innominate artery (Fig. 1).

Dr. John H. Moon: You said her heart was normal considering her age. What does that mean?

Dr. Neal: As people get older, the heart shadow becomes larger, even without hypertension.

The lateral cubitus film did not reflect any free air and there were very few air-

fluid levels in the small bowel. A laminated gallstone in the gallbladder area and calcification of the arteriosclerotic, tortuous



Fig. 1. There is marked arteriosclerotic change in the aorta. The bones appear poorly mineralized. A mass at the right apex causes deviation of the trachea to the left.

splenic artery were noted. There is calcification present in the pelvic vessels. There is fairly good sized, distended, air-filled stomach on the AP abdomen film. Looking through the midportion of the abdomen, we note gas in a rather generalized distribution through the small bowel. It is notable that the colon is relatively free of this overall distribution of gas. If we look at these opposed loops, the degree of tissue between them does not reflect fluid as we might see with peritonitis. Also, it does not suggest the edema that you might anticipate with venous obstruction (Fig. 2).

The degree of filling of the stomach and the small bowel, radiographically, makes me think of an ileus as the best possibility.

Dr. Rittenbury: Did you see any calcification around the kidneys? Is the generalized demineralization excessive or compatible with her age?

Dr. Neal: I see no calcification about the kidneys. The demineralization is compatible with her age.

Dr. Rittenbury: In considering this patient, particularly in view of her x-ray findings as described in the protocol before hearing Dr. Neal's interpretation, we must think of acute hyperparathyroidism. There are several features that go with this. The first is her history of nausea and vomiting; this occurs in up to 80 percent of people with hyperparathyroidism. Compatible with this disease are her weight loss, her confusion, back pain—if you consider this bone pain, and, possibly, the amylase of 204 units, remembering that pancreatitis will occur in a certain number of people with



Fig. 2. The small bowel loops are dilated but with little space between them as would be seen in peritonitis. The colon is spared. A gallstone is evident as well as a sclerotic aorta and tortuous splenic artery. These findings were interpreted as ileus.

hyperparathyroidism. The mediastinal mass could be a parathyroid adenoma. There are certain things that go against this diagnosis in

her history. First is her age. Of patients with hyperparathyroidism, with acute symptoms, as this patient apparently showed, it is the extremely rare one that develops the disease after age 60. In addition, most of these patients have lethargy; they may have weakness and they may have other bone pain with the demineralization. Over half of them show a neck mass; this lady did not have one. Two other striking findings found in acute hyperparathyroidism are a change in the QT intervals on electrocardiogram which apparently was not present here, and also hypokalemia. Hyperparathyroidism has to be considered, but I think we can rule it out.

We must consider the mass which is described in the protocol as a lung mass with encroachment upon the superior mediastinum. In this position and as described by radiologists, I think enlargement or buckling or aneurysm of the innominate artery would be the most probable cause here. In considering anterior mediastinal masses, you find that around 15 percent of all mediastinal masses will be composed of thyroid or parathyroid tumors or cysts, thymomas, teratoid dermoid cysts. In the superior mediastinum lymphomas would be frequent anteriorly, while in the posterior and lateral mediastinum you have to think of neurogenic tumors. Bronchogenic carcinoma involving the mediastinum to appear as a mediastinal mass rather than as a pulmonary mass is an unlikely possibility. There are two other tissues that could cause masses in this area. One is the pleura. Pleural mesotheliomas are being diagnosed with greater frequency. Most of these patients, however, do have symptoms of cough and chest pain; most of them have physical signs as in the chest and most of them have pleural effusion. This lady had none of these. Pulmonary tumors, of course, enter again into the differential diagnosis. In the female, the adenocarcinoma accounts for 15 percent of all carcinomas of the lung. It is usually peripherally located. A vascular defect appears to be the correct diagnosis in our patient, however.

We go next to the differential diagnosis of a patient with anorexia and abdominal pain for a year. To me these are fairly nonspecific because I think that so many patients that we see have both symptoms in this age group; therefore, I am going to rather discount these and dwell more upon what appears to me to be her chief findings.

This is a patient who had eight days of nausea, vomiting and diarrhea; two days prior to coming into the hospital the diarrhea became bloody. What are the causes of bloody diarrhea? We can think of regional enteritis; against this is her age of 72. These patients usually do not have this predominance of nausea and vomiting although they can have chronic abdominal pain. In addition, most of the bleeding is not severe or as profuse as apparently this case was. Colitis does not usually have its onset at this late stage of life, and she has no history of colon disease. Ordinarily she would have had symptoms at an earlier age and have recurrent pain. This may be the cause of her chronic abdominal pain, however. In addition, about 85 percent of all people with colitis will have some involvement of the rectal segment. I think we can rule out colitis, as it was not seen on the proctoscopic examination. Diverticulitis or ruptured diverticulum will give a wide range of clinical manifestations but they don't really fit this particular picture. Usually 50-60 percent of the people over 60 years of age will have some diverticuli that can be shown by barium enema on x-ray. Gross rectal bleeding is not frequent with diverticulitis. Usually there is not this amount of diarrhea. With the diagnosis of diverticulitis, the findings are usually more localized and if there is a perforation you would expect to see a more localized area of peritonitis. We usually think of diverticulosis as more likely causing massive rectal bleeding than diverticulitis. When people with diverticulosis do have rectal bleeding it is usually

without diarrhea. Also, they do not have the prodromal symptoms or signs that this patient had.

We have to consider the infectious diseases. I think first of Salmonella typhosa. This doesn't fit, as those patients have cough, intermittent fever, chills, headaches, constipation, drowsiness, rash and splenomegaly. I think the clinical onset is not typical of Salmonella typhosa, the Salmonella food poisonings or the staph food poisonings. Usually with these, a group of people are involved, the onset of the diarrhea and vomiting is explosive and it usually lasts from one to two or three days, with fever. In Shigellosis, 25 percent will have blood in their stools; however, this is an abrupt, toxic disease and not this prolonged. Amebic dysentery is usually more insidious and yields more mucus and less blood. This lady could have had a viral gastroenteritis. I am not really sure what the term means except that to me it represents a nonspecific gastroenteritis that lasts one, two or three days and is associated with some fever, muscular pains and aches. The diarrhea is rarely bloody. It is possible that she started with her first nausea and vomiting and diarrhea due to a viral enteritis. Staphylococcal enteritis has to be considered but it is usually a more toxic process.

The next group of diseases that can cause these symptoms are the vascular diseases of the small and large bowel. There has been much discussion concerning whether or not an arterial or venous mesenteric occlusion can be diagnosed differentially without laparotomy or autopsy. Without belaboring the point, I don't think you can differentiate the lesion.

Myocardial infarction is frequently the cause of mesenteric emboli. If such were the case we would expect a more acute process and also evidence of other emboli. Necrotizing arteriolitis, isolated or associated with rheumatoid arthritis, lupus or periarteritis nodosa, can cause occlusion of large or small mesenteric arteries, causing hemorrhage necrosis of the bowel. She has no other evidence of arteritis, that I could find.

Druker[1] has recently described a syndrome of hemorrhagic necrosis of the bowel without gross vascular occlusion. This can be associated with dehydration, heart disease, myocardial infarction, shock or other conditions that may decrease the circulating volume in a patient who has arteriosclerosis and who probably has a partial or relative occlusion to the superior mesenteric artery. These patients usually have nausea and distention, hypoperistalsis or ileus. The patient may have an elevated or only slightly elevated temperature and leukocytosis. I think the findings of hemorrhagic necrosis of the intestine are certainly compatible with the present patient's disease course. I would think that her course would be compatible with the onset of a gastroenteritis followed by nausea and vomiting, dehydration and hemorrhagic necrosis or fine vascular occlusion with or without large vessel occlusion of the small bowel.

Dr. Moon: I didn't quite understand your distinction between the diverticulosis, diverticulitis and the bleeding in diverticulosis. I always assumed that bleeding from diverticula was the result of infection in the diverticulum. It is not right?

Dr. Rittenbury: Dr. Nakonecza can give the morphological differentiation better than I can, but you can have bleeding in both instances. However, the grossly bloody diarrhea or gross rectal bleeding is associated with the "osis", this being the presence of diverticuli rather than a true clinical diverticulitis. Now, if you want to say that we are talking about microscopic inflammatory response and erosion of a vessel in a diverticulum, then, of course, one would have to have "itis". I am thinking more of a clinical than a morphological diagnosis.

Dr. Moon: What were the thoughts of the senior students?

Dr. Ferlazzo (Senior Student): The seniors favored various diagnoses including carcinoma of the pancreas, mesenteric vascular occlusion, pulmonary tuberculosis with secondary ileocecal involvement and carcinoma of the colon.

Ward Diagnosis:

1. *Mesenteric artery thrombosis*
2. *? mesenteric venous thrombosis.*
3. *? small bowel volvulus.*

Dr. Rittenbury's Diagnosis:

Small bowel necrosis, secondary to:

1. *atherosclerosis of mesenteric arterial tree*
2. *congestive heart failure, and*
3. *viral gastroenteritis.*

Pathological Discussion

Dr. Irene Nakoneczna: Laparotomy disclosed gangrene of a segment of the small intestine, approximately four feet in length. There was no fibrous band or volvulus. The major branches of the superior mesenteric artery were pulsating, however, no distal pulsations could be felt. Apparently there was marked peritoneal contamination. The gangrenous segment of the bowel was resected and the peritoneal cavity was irrigated.

The patient remained hypotensive throughout the procedure and she developed irreversible cardiac arrest shortly after reaching the recovery room.

At autopsy, the patient was 4 feet 9 inches tall and weighed 80-85 pounds. She was malnourished and generally dehydrated but there was mild pitting edema of the ankles.

The pleural cavities contained clear-yellow fluid; there were 300 cc. on the right side and 100 cc. on the left. The lungs weighed 550 grams and showed diffuse atelectasis. The tracheobronchial tree was filled with gastric material from terminal agonal aspiration. An ill-defined density was present in the right upper lobe. The upper one-third of the right pleural cavity was obliterated by dense fibrous adhesions. Microscopically the lungs showed diffuse atelectasis, interstitial fibrosis and arteriosclerosis of pulmonary vessels. There were areas of chronic interstitial pneumonia alternating with broad bands of fibrous tissue and scattered non-caseating granulomas, probably tuberculous in origin, in the upper lobe of the right lung. Similar non-caseating granulomas were found in the adjacent bronchial lymph nodes. Special stains for tubercle bacilli and fungi were negative.

The heart weighed 300 grams, slightly hypertrophic for a person her size. The coronary arteries were narrowed and the myocardium showed mild fibrosis. The liver showed the changes that follow passive congestion.

The small arteries and arterioles showed marked hyalinization with narrowing of their lumen indicating hypertension. The grossly enlarged kidneys showed degenerative changes commonly seen following prolonged periods of poor arterial perfusion.

The peritoneal cavity contained approximately 300 ml. of serosanguinous fluid. The GI tract was filled with dark-brown coffee ground-like material. The recent small bowel anastomosis was approximately 9 cm. distance from the ileo-cecal valve and about 125 cm. from the ligament of Tritz. The infarcted segment of bowel removed was 127 cm. in length. The entire small bowel and adjacent mesentery was markedly congested grossly and showed diffuse hemorrhagic areas plus areas of necrosis (Fig. 3) close to the surgical anastomosis. Microscopic examination of the surgical and autopsy material showed diffuse necrosis, particularly of the intestinal mucosa. Adjacent areas revealed mucosal atrophy suggestive of chronic ischemia (Fig. 4). There was also an acute inflammation in the mesentery and adjacent peritoneum.

Now, the important thing is that examination of the mesenteric vessels did not disclose recent thrombus; however, there

was diffuse sclerosis of vessels. There was a partial occlusion of the superior mesenteric artery near its origin as well as severe sclerosis of branches of the mesenteric.

You will remember that superimposed upon the effect of this limited vascular tree was the progressive heart failure secondary to arteriosclerotic hypertensive cardiovascular and chronic pulmonary disease. We believe that the marginal blood flow and ischemia of the bowel during episodes of congestive heart failure account for the intestinal trouble which this patient had over a period of one year. It has been shown[2] that poor perfusion of the intestine can cause post-prandial pain that induces the patient to eat less. The poor perfusion can also cause intestinal mucosal atrophy which impairs food absorption.

With a more severe fall in cardiac output, such as accompanies congestive heart failure, shock and myocardial infarction, the body selectively redistributes its depleted vascular volume. The vital organs such as brain and heart benefit from the re-allocation of blood, at the expense of the gastrointestinal tract, kidneys and extremities. Ischemia stimulates the smooth muscle of the bowel wall to go into spasm, which tends further to interfere with blood supply to the intestinal wall.[3] Vasospasm here appears to be a physiologic compensation for a reduced cardiac output, but a compensation that can have disastrous results.

Compromise of the vessels of the gut presents a complication not associated with other ogans except perhaps the skin. This is the abundant bacteria in the tract. As ischemia progresses, the bowel flora invade the intestinal wall and their toxins contribute to the morbid process.



Fig. 3. In areas of early gangrene, only an outline of necrotic mucosa remains. Submucosal edema and vascular congestion are prominent (H & E stain, 40X)

In summary, this patient had a chronically poor perfusion of her intestinal tract due to mesenteric arteriosclerosis and her congestive heart failure. The terminal acute episode was probably precipitated by more severe heart failure. This led to mucosal ulceration, intestinal fluid loss, bacterial invasion, further reduced intestinal vascular

flow, and gangrene of the small bowel and death.

Dr. Page Hudson: (Associate Professor in Surgical Pathology)

There are two problems here with terminology. The term "intestinal angina" has been used for the abdominal pain associated with lead poisoning, luetic crisis, porphyria, and abdominal epilepsy as well as the two different entities that are known as "superior mesenteric artery syndrome." This latter is used by some to refer to the intermittent obstruction of the duodenum by the superior mesenteric artery. The term means to others the effects of relatively inadequate perfusion through the mesenteric arteries. Such is the case under discussion today. Dr. Rittenbury certainly did not open the wrong door.

Dr. Rittenbury: I want to emphasize that relatively distinct signs and symptoms of vascular disease of the intestine may develop months or years before frank gangrene or death, and that there is distinct surgical, as opposed to medical, therapy for many of these patients.[4]

Final Diagnosis:

1. *Gangrene of small intestine, secondary to arteriosclerotic mesenteric artery insufficiency combined with coronary artery disease and cardiac failure.*
2. *Renal tubular necrosis, acute.*
3. *Chronic, organizing pneumonia, focal.*

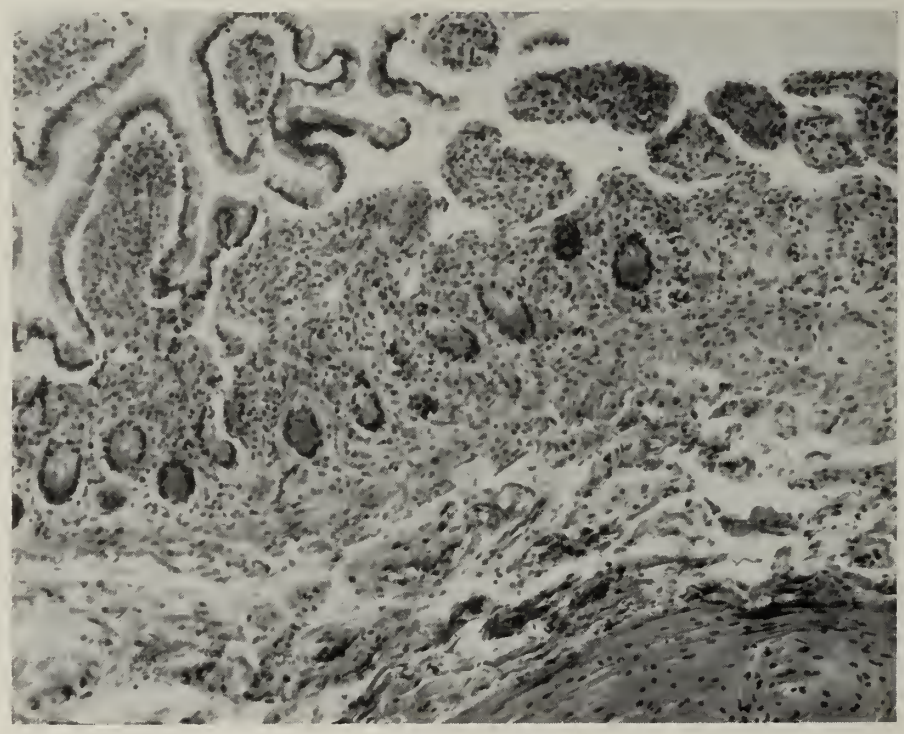


Fig. 4. Atrophy of the mucosa (particularly on right) was present in non-gangrenous areas of small intestine, reflecting vascular insufficiency (H & E stain, 100 X)

The illustrations were prepared through the facilities of the Department of Visual Education, Mr. Melvin Shaffer, Director.

Please address reprint requests to Dr. Page Hudson, P. O. Box 871, Medical College of Virginia, Richmond, Virginia 23219.



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In the hypotonic duodenograph[1,2] reproduced above, the gastrointestinal tract was relaxed with Pro-Banthine. The duodenum was intubated. Pro-Banthine in a dose of 60 mg. intramuscularly was used to assure prompt aperistalsis and double-contrast visualization was achieved with ordinary barium and air.

The same pharmacologic efficiency has proved of pronounced value in such conditions as: *peptic ulcer, pylorospasm, biliary dyskinesia, functional hypermotility and irritable colon.*

Contraindications: Glaucoma or severe cardiac disease.

Precautions: Since varying degrees of urinary hesitancy may occur in elderly males with prostatic hypertrophy, this should be watched for in such patients until they have gained some experience with the drug. Although never reported, theoretically a curare-like action may occur with possible loss of voluntary muscle control. Such patients should receive prompt and continuing artificial respiration until the drug effect has been exhausted.

Side Effects: The more common side effects, in order of incidence, are xerostomia, mydriasis, hesitancy of urination and gastric fullness.

Dosage: The maximal tolerated dosage is usually the most effective. For most *adult* patients this will be four to six 15-mg. tablets daily in divided doses. In severe conditions as many as two tablets four to six times daily may be required. Pro-Banthine (brand of propantheline bromide) is supplied as tablets of 15 mg., as prolonged-acting tablets of 30 mg. and, for parenteral use, as serum-type vials of 30 mg. The parenteral dose should be adjusted to the patient's requirement and may be up to 30 mg. or more every six hours, intramuscularly or intravenously.

(1) Bilbao, M. K.; Frische, L. H.; Rösch, J., and Dotter, C. T.: Hypotonic Duodenography, Scientific Exhibit, Radiological Society of North America, Chicago, Nov. 27—Dec. 2, 1966.

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Dunga Gin

You may talk of sex and whisky
When you're feeling good and frisky,
An' you're sitting at the frat house drinking 'round.
But when it comes to enteritis
That's something else entirely,
An' deservin' of some thought more profound.
'Twas about a year ago
Between the hours of two and four,
When the first good brothers began to regurgitate.
With some explosive diarrhea
For which there is no panacea,
They were convinced it must have been something they ate.
Then around six o'clock
To the E.R. they did flock,
In groups of two, or three, or more.
Those seen vomiting in the gutter
Were fast becoming *persona non grata,*
That night they were admitted to South Four.
They were sick, sick, sick,
Hurry, double quick, the cuspidor.
For 'tis far from academic,
In an enteritis epidemic,
As to who next gets to use the lavator.
Holding forth at our institution
Searching vainly for a solution,
Were Porter, Hollingsworth, Connally, and McGraw.
"Was it staph toxin, or shigella,
Perhaps clostridium, or salmonella?"
The answer was not forthcoming to them all.
So rather than take a chance
To the wards they advanced,
Until an even dozen were admitted.
At this point they did perceive
That this was no lethal disease,
And their fears, soon thereafter, remitted.
Twelve hours later they were all doing well,
It could have been staph or salmonell.
One year later 'twould be a delusion
To say that we've come to a better conclusion.

Henry K. Butler, M.D.

Editor's Note: Dr. Butler is a Fellow in Infectious Disease at the University of Virginia Hospital.

One year ago an outbreak of acute gastroenteritis occurred in a fraternity house at the University of Virginia. Despite an extensive investigation, the cause of the epidemic was not found. With apologies to Rudyard Kipling, Dr. Butler has submitted the above poem. It is dedicated to the 54 stricken fraternity men and the intrepid house staff who cared for them.

Correspondence . . . .

The Unbalanced Equation

To the Editor:

In an editorial in the December 1967, issue of The Virginia Medical Monthly, Dr. Monford D. Custer, Jr., recommends surgical resection of the thyroid gland for all patients with hyperthyroidism who have "significant goiter". Some comments are made about iodine deficient goiter and nodular goiter which do not seem to bear directly on the treatment of hyperthyroidism. However, as I understand this editorial, the author's recommendation is based primarily on his feeling that in hyperthyroidism the goiter" . . . seldom recedes with management limited strictly to medical measures." He therefore suggests that non-surgical therapy will result in the persistence of an unsightly goiter which will probably also produce symptoms by mechanical compression.

I believe that Dr. Custer is mistaken in his contention that goiters seldom recede following ^{131}I therapy for hyperthyroidism. In my experience, the goiter always decreases in size following ^{131}I therapy for Graves disease. Most glands return to approximately normal size and often to subnormal size. Even very large goiters will usually return to approximately normal size, although some of these will remain slightly enlarged to palpation. However, I have not yet encountered a patient treated adequately with ^{131}I in whom the gland remained large enough to present a cosmetic problem or to cause compression symptoms.

The cause of Graves disease is not known and none of the presently available therapies is ideal. The physician may have valid reasons for choosing any one of these. Both surgical and non-surgical methods of treatment regularly give satisfactory cosmetic results so that this does not seem to be a significant factor in the choice of therapy in a great majority of cases.

Halcott T. Haden, M.D.
Chief, Hematology Section
& Radioisotope Laboratory

December 15, 1967

Veterans Administration Hospital
Richmond, Virginia 23225

Survival After Prolonged Coma

To the Editor:

Dear Sir:

The ability of physicians to maintain life for very long periods in the unconscious patient raises the question as to how long such skills should be deployed. As physicians we are eager to promote the recovery of everyone who can do so. In order to deprive no one of his chances on this score it is relevant to know the longest periods of coma which have been followed by useful survival.

A committee of the Massachusetts General Hospital is studying our own records and the world literature to determine pertinent features in all patients who, *despite coma for over 5 weeks,* have made a useful recovery. We think it is vital not to overlook any well documented patient in this category. We should be grateful if any reader of this journal would draw our attention to any case published under a title which is not indicative of survival after prolonged coma. We are also eager to receive accounts of such cases as yet unreported. A publication incorporating our own and others' data is planned.

We should be grateful if you would publish this letter in your journal either in a

section for correspondence, as a special brief communication, or in any other fashion you see fit.

Sincerely yours,

WILLIAM S. SWEET, M.D., D.Sc.
Chief, Neurosurgical Service Chairman, Committee on Management of the Unconscious Patient

January 24, 1968

Massachusetts General Hospital
Boston, Massachusetts 02114

The Circadian Rhythm in Treatment

TO THE EDITOR:

The Editorial on The Physiological Clock by Dr. Robert Edgar Mitchell, Jr., is an interesting condensation on research done on this subject. In the Virginia Medical Monthly for October 1960 (Vol. 87 p. 578), in the Journal of the American Medical Association for Dec. 10, 1960 (Vol. 174, p. 19940) and in the Pennsylvania Medical Journal, for February 1966 (Vol. 69, page 63), I have suggested the following theory:

The importance of the seven-day-periodicity of inflammatory disease is stressed in Leviticus, chap. 13, in Numbers, ch. 12, v. 14, etc. The Hebrew phrase MAKOH SHEVA in Leviticus, ch. 26, v. 21 points towards the neoplastic and degenerative conditions seen to develop following interruption, by means of antibiotics, antihistaminics and corticosteroids, of this seven-day-periodicity.

The data published on page 25 of Smoking and Health (Department of Health, Education and Welfare, 1964) appeared to corroborate my theory. Tobacco has been smoked since (long before) Columbus discovered America; the increase in the indidence of neoplastic and degenerative disease has risen sharply since the newer drugs have appeared.

Whether it was post hoc or propter hoc, since the appearance of my last article the Food and Drugs Administration has ordered a reevaluation of all the drugs that have appeared on the market since 1938, and I sincerely hope that this reevaluation will serve a useful purpose. In the early 1931, while I was a Hilfsarzt at the Psychiatrche und Nervenklinik der Universitaet Wuerzburg we have decided to try the prolonged sleep treatment on our patients. It took several days to show that while this treatment seemed to help some, it was nonphysiologic and even dangerous. Following this experiment, it appeared to me that as a rule, sleep should be useful only at night. Patients were then given hypnotics only at bedtime and the ward which was filled with restless and unmanageable patients at once became quiet. This was the day-and-night periodicity in treatment which is now called the circadian rhythm. In private practice I have used phenobarbital 0.1 Gm one or two tablets to be given at bedtime. The results were good especially in depressed states and some schizophrenias. A schizophreniac which has been in remission for several years after spending some time in a state hospital, became restless, sleepless and unmanageable again. He was given phenobarbital at bedtime and within one month he improved to such an extent that hospitalization was no more believed to be necessary. Improvement continues without phenobarbital. His brother who has spent some time in the hospital with an abortive attack of schizophrenia, and who has been discharged with a good supply of tranquilizers, had the tranquilizers discontinued and was given phenobarbital, 0.1 Gm. at bedtime. He never had to return to the hospital and has been taking now phenobarbital for several years without any signs of recurrence of the psychosis. A 70 year old man had developed a depressed state and was suicidal. He was sent to the hospital and returned with tranquilizers. The tranquilizers were discontinued and phenobarbital 0.2 Gm hs were given to him. He reacted well to the treatment and improvement continued for one year. Recurrence of depression took place

when he stopped taking the phenobarbital tablets and he had to be hospitalized again. Upon his return from the hospital the tranquilizers were discontinued again and he was again given phenobarbital tablets to be taken hs. He continues to act normally now three years afterwards. These are only some of the cases seen. Phenobarbital 0.1 Gm or 0.2 Gm hs was found to be useful in the treatment of grand mal epilepsy. All the cases of grand mal epilepsy improved and in most of the cases no convulsions were seen to occur when phenobarbital was given only at bedtime. This was going on for 28 years and I am pretty well satisfied with the results obtained. My experiences with coronary insufficiency are rather limited but when aminophylline 0.1 Gm was given to them at bedtime supralingually (sublingual treatment with aminophylline is not practicable because of the injury it causes to the mucosa), the progress of the condition seemed to be better than when no hs treatment was used. Ankle edema seemed to improve in refractory cases when aminophylline was given hs, and when it was dissolved in the mouth.

These experiences appear to suggest that the circadian rhythm can be very useful in the treatment of psychoses, epilepsy (grand mal), cardiam edema, and coronary insufficiency.

Leo I. Hallay, M.D.
Fort Blackmore, Va. 24250

Search Continues for Health Care Solutions

The AMA News, a publication of the American Medical Association, said in an editorial that the AMA will "continue to seek solutions to the problems of health care for the poor" despite "harassment, even from those it is trying to help."

Commenting on the recent AMA-sponsored National Conference on Health Care for the Poor, The AMA News said that the conference drew experts with a wide variety of viewpoints "to discuss and search for answers to a national problem" and "the results were educational to the AMA and to those invited to participate in the conference."

"However, an apparently coordinated, well-planned attack on the AMA and the conference succeeded in diverting the attention of many, including the news media, from the discussions of health care for the poor. How that was done also proved educational.

"News releases issued before the conference began got headlines by rehashing old arguments against organized medicine. During the conference's final day, a placard-carrying group of about 100 people representing a Chicago organization marched into the session demanding to be heard. When their representatives were given the floor, they mistakenly criticized the conference for including 'no voice from the poor' and for attempting 'to silence the anguished cries of the poor.'

"Even before the conference concluded, still another group issued a last statement criticizing the conference as being unproductive.

"The kind of harassment to which the AMA and its conference were subjected consists of motion, of noise, and of rigid expressions of opinion. The American Medical Association, of course, is not the only one to be subjected to the kind of grasping for attention at almost any price which seems to be a part of today's social attitude. Dissent and disagreement for many have come to mean a flaunting of manners and of reason."

Diagnostic Laboratory Medicine . . . .

M. J. ALLISON, Ph.D.
H. P. DALTON, Ph.D.
M. R. ESCOBAR, Ph.D.

The Frequency of Salmonella and Shigella Infections in the Richmond Area

Among the enteric pathogens of man, Shigella and Salmonella species have a universal distribution as a causative agent of disease. Since man is the sole natural source of Shigella organisms, the number of clinical cases will fluctuate with the level of sanitary practices in a given area. Similarly Salmonellosis may be caused by such practices. In general, however, the Salmonella organisms are involved in non-human disease with infection in man being only accidental. In recent years there have been a number of reports that Shigellosis is declining and Salmonellosis is on the increase. Our present 12 year report (1956-1967) bears this out. Of the 416 cases of Shigellosis there were 159 in the last six years and 257 in the first six years. Salmonellosis was the reverse; out of 343 cases, 195 were in the last six years and 148 were in the first six years.

Considering the 416 cases of Shigella, in all instances the organism was isolated from stool specimens and represented three of the four serological groups. Two hundred and seventy-three isolations were group B, S. *flexneri*, one isolation group C, S. *boydii*, and 142 isolations group D, S. *sonnei*. This is equivalent to 10.2 cases of Shigellosis per 10,000 hospital admissions.

The frequency of Salmonella infections in the Medical College of Virginia is 8.5 cases per 10,000 admissions. The 343 cases of Salmonella, represented by 34 different species, are listed in Table I. The principle cause of Salmonellosis in the Richmond area is *Salmonella typhimurium*. This is the leading cause of Salmonellosis in many parts of the world. The frequency of other species in causing human disease varies considerably with eating habits and environment. Nearly all species are able to produce epidemics under the proper circumstances. Disease with any given species may manifest itself either as a rather mild enteritis, commonly described as food poisoning or as a severe systemic disease. The bulk of our cases was simple food poisoning with 64% of the isolations coming from the stool. Eighteen per cent were isolated from the blood with another 18% coming from such miscellaneous specimens as urine, aneurysm, sputum, gall bladder, spinal fluid, joints, peritoneal fluid and wounds.

Typhoid fever was more common in the 1956-1961 period with 14 cases; in the last six years there were only five cases. This is true also for the virulent organism S. *choleraesuis* (Kunzendorf) with 12 cases in the first six years and three in the last six years. This species seems to show a special predilection for the production of mycotic aneurysms since four of the 15 cases of Salmonellosis caused by this species were accompanied by mycotic aneurysms. Only one additional Salmonella mycotic aneurysm was seen and that in a case due to S. *enteritidis*. This latter species has increased in frequency in cases of Salmonellosis from four cases in the first six years to 31 in the second six years.

There have been seven newcomers, as far as species are concerned, to the area in recent years: ohio, thompson, livingstone, berta, manchester, remo and one untypable. On the other hand, we have lost some species in the area in recent years that were previously found here. These are reading, paratyphi B, kentucky, javiana, minnesota and worthington. The loss and addition of

species such as these are probably not of any great epidemiological significance as they are low frequency species and will probably appear and disappear in future years as well.

In conclusion it would appear that Salmonellosis and Shigellosis, as seen in our institution, is following the general pattern reported in other parts of the United States.

SALMONELLOSIS AND SHIGELLOSIS
MEDICAL COLLEGE OF VIRGINIA
1956-1967

Shigella	*1956-61*	*1962-67*	*Total*
A	0	0	0
B	188	85	273
C	0	1	1
D	69	73	142
Total	257	159	416

Salmonella	*1956-61*	*1962-67*	*Total*
Group B			
typhimurium	54	58	112
chester	2	4	6
reading	3	0	3
st. paul	2	1	3
derby	7	3	10
paratyphi B	1	0	1
heidelberg	6	14	20
indiana	4	1	5
remo	0	1	1
untypable	0	1	1
Total	79	83	162
Group C			
choleraesuis (Kunzendorf)	12	3	15
oranienburg	6	1	7
infantis	7	28	35
montevideo	4	8	12
tennessee	1	1	2
bareilly	2	3	5
untypable	2	1	3
litchfield	5	2	7
kentucky	1	0	1
manhattan	1	1	1
newport	3	7	10
muenchen	1	1	2
blockley	1	6	7
manchester	0	1	1
ohio	0	2	2
thompson	0	4	4
livingstone	0	1	1
Total	46	70	116

Salmonella	*1956-61*	*1962-67*	*Total*
Group D			
typhosa	14	5	19
javiana	2	0	2
enteritidis	4	31	35
berta	0	2	2
Total	20	38	58
Group E			
anatum	1	4	5
minnesota	1	0	1
Total	2	4	6
Ungrouped worthington	1	0	1
Salmonella Total	148	195	343

Division of Clinical Pathology
Medical College of Virginia
Richmond, Virginia 23219

Public Health . . . .

MACK I. SHANHOLTZ, M.D.
State Health Commissioner of Virginia

Changes in Health Department Facilities Attributable to Modern Treatment of Tuberculosis

Since the introduction and extensive use of antimicrobial agents effective against the tubercle bacillus, many definite trends have been observed. In the last few years the number of tuberculosis deaths, the number of sanatorium patients, and the average length of sanatorium stay have fallen markedly. In 1957 there were 334 tuberculosis deaths with a rate of 8.6 per 100,000; in 1966 there were 174 deaths with a rate of 3.8 per 100,000. In 1960 there were almost 1,000 patients in sanatoria at the end of the year; in 1967 there were only 486. The average length of stay in sanatoria was 353 days in 1955, and in 1967 it was down to 191 days.

Conversely, shorter hospitalization and more home treatment have combined to greatly increase the patient load at our 34 regional chest clinics. In 1958 there were 1,796 patients seen in regional clinics while in 1967 the clinics served 4,840 patients. In 1955 the success of isoniazid administered prophylactically in a United States Public Health Service trial ushered in a new age of prevention, the age of chemoprophylaxis. Chemoprophylaxis undoubtedly has helped cut down on our sanatoria population. Moreover, in recent years it has become increasingly more acceptable to treat early non-communicable cases in the home.

Owing principally to the factors mentioned above, the Health Department was able to close out its Piedmont Sanatorium operation July 1, 1967. (During our phase-out period, the Department of Mental Hygiene and Hospitals moved geriatric mental patients into Piedmont and incorporated the hospital into its system on July 1, 1967.) At the time of this writing, admissions to Ennion G. Williams Sanatorium are about to be "shut off", and on July 1, 1968, it will "close out" it operation as a sanatorium, leaving Blue Ridge and Catawba as the only remaining sanatoria in the State. The State Health Department will retain its present regional chest clinic on the ground floor of the Ennion G. Williams Hospital, and on or about July 1, 1968, it is planned to move the Medical College of Virginia unit of the State Health Department's Bureau of Alcohol Studies and Rehabilitation from its present location in the Memorial Hospital (MCV South) to the ninth and tenth floors of Ennion G. Williams Hospital (MCV North), which we intend to modify and refurbish during the Spring months of 1968. The contemplated new larger facilities for the alcoholic program would provide for the inpatient accommodation of up to 50 patients at any one time in contrast to a limitation of 12 patients in the present unit. There would also be an increase in the activities of the outpatient clinic. As in the past, the MCV unit of the Bureau of Alcohol Studies and Rehabilitation would continue to work closely with all of the other clinics and services of the Bureau and intends to develop further its functions in teaching, research, and other services to the people of Virginia.

In the planned new unit for alcoholics, every effort would be made to identify and accept patients for therapy as early as possible in the course of their illness and as soon as possible after they or their families ask for help. Provision would be made in the new unit for greatly expanded outpatient clinic facilities which would permit efficient

utilization of a much larger outpatient staff. This would result in prompt appointments for all referrals which has not been possible in the past.

As the contemplated new unit is developed, it is intended to provide, as a part of the comprehensive program, a specialized area for an acute crisis emergency service. The function of this service would be to handle acute detoxification problems and the duration of stay in this service would be studied as well as appropriate staffing and operation. It is intended that this acute service would work in cooperation with the general emergency services of the hospital. As experience is gained in operating the planned acute crisis emergency service it is hoped that wise decisions can be made as to which patients can most effectively be helped in this way and exactly how their subsequent course of therapy can be planned.

Monthly Report of Bureau of Communicable Disease Control

	January 1968	January 1967
Brucellosis	0	4
Diphtheria	0	0
Hepatitis	44	48
Meningitis (Aseptic)	0	1
Meningococcal Infections	6	5
Poliomyelitis	0	0
Rocky Mt. Spotted Fever	2	0
Rubella	20	18
Rubeola	19	199
Streptococcal Sore Throat (including Scarlet)	1567	1577
Tularemia	0	0
Typhoid Fever	0	0
Rabies (in animals)	17	16
Venereal Diseases		
Syphilis	176	170
Gonorrhea	873	734
Other	0	0

Sources of sinus headache



REGION OF SENSATION

FRONTAL SINUS—headache localized over the frontal region, commonly beginning in the morning. Eyes painful to touch, may be slight swelling over eyes, puffy eyelids.
SPHENOID SINUS—most difficult to diagnose. Headache or sense of pressure over vertex. May be pain in ear or mastoid region.



MAXILLARY SINUS—headache and tenderness under eyes at top of cheekbones, usually starting in early afternoon. Possible swelling of cheek. Pain in jaw and back teeth.
ETHMOID SINUS—headache, tightness in upper part of bridge of nose and well down its sides. Pain, deep-seated behind the eyes and in inner corners of the eyes.



TURBINATES—pain along medial and lateral walls of the inside of the nose. Headache in frontal region, along supraorbital ridge, a feeling of fullness in the head.

Source of symptomatic relief

SINUTAB



Sinus headache is often a reflection of congestion in the nasal mucosa. The pain that results in the various regions of the head may help in determining the particular structure(s) responsible. The Sinutab formula is specifically designed for symptomatic relief of sinus headache and nasal congestion. Sinutab *not only* **provides an effective decongestant** — to reduce mucosal engorgement and facilitate freer breathing — *and* **an antihistamine** — to help reduce edema and excessive secretions — but it *also* provides, in contrast to other leading decongestants, **two analgesics** for immediate relief of pain and discomfort.

And, too, the Sinutab dosage schedule of 2 tablets, *q.* 4 *h.* permits controlled and continuing analgesic relief.

Adverse Reactions: Epigastric distress, drowsiness, dizziness, insomnia and nervousness.

Precautions: Instruct patients not to drive or operate machinery if drowsiness occurs. Use with caution in patients with thyroid disease, heart disease, hypertension, diabetes or kidney disease. Excessive dosage or prolonged use may cause kidney damage.

Dosage: Adults—2 tablets every 4 hours.

Each tablet contains 150 mg. acetaminophen, 150 mg. phenacetin, 25 mg. phenylpropanolamine HCl, and 22 mg. phenyltoloxamine citrate.

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Mental Health . . . .

R. W. GARNETT, JR., M.D.

Community Psychiatry or Community Mental Health

Community Psychiatry is that branch or aspect of Psychiatry that is concerned with the mental health of the entire population of a community and is related to clinical psychiatry in the same way that Public Health is related to clinical medicine. Community Psychiatry deals with prevention, diagnosis, treatment, and rehabilitation of mental disorder in all the range of its manifestations, as it occurs in a whole population, rather than focusing down on individual pathology. It involves planning and administration to provide a comprehensive range of programs and services designed to reach a maximum number of people for the optimum care of preventative, therapeutic and rehabilitative needs consistent with social, economic and political reality.

Community Mental Health encompasses Community Psychiatry which includes clinical psychiatry, and also involves all other mental health professionals and care taking agencies such as social workers, psychologists, social scientists, teachers, ministers, police, politicians, administrators, lawyers, judges, public health officials and nurses, practicing physicians, rehabilitation counselors, volunteer mental health workers, etc.

Community Psychiatry is also known as Social Psychiatry, Public Health Psychiatry and Public Mental Health.

All this obviously implies that the psychiatrists and other mental health professionals must learn to work in new roles, in new settings and in new and different patterns and on other levels than in the traditional ones. These new roles and patterns of behavior for psychiatrists have not been sought or imposed by a governmental bureaucracy, but reflect the changing expectations of society itself—the demands of the public and the trends affecting all of medicine, particularly in the patterns of medical care—the increasing emphasis on prevention, early treatment, and rehabilitation, the increasing demand for adequate health services by and for all segments of the population, the growing network of care giving agencies and institutions of society demanding better coordination and organization.

It has been a popular joke or a public scandal, depending on the frame of reference, that adequate psychiatric services and facilities were available only to the affluent. In a study by William Ryan in Boston, he found that 70% of patients in private outpatient psychiatric treatment came from 7% of the population, and that nearly half of those in the community identified as emotionally disturbed never get to any kind of psychiatric, medical or social resource, public or private. One third of those identified and referred to some care giving agency never got there. In other words, only a sixth of those needing help received any, even in Boston.

Many other studies have borne out the same theme. I will not bore you with a lot of statistics. Someone has said that if all statistics were laid side by side—it would be a good thing. Suffice it to say that a few good community studies have been done —the Midtown Manhattan Study, the Sterling County Study in Nova Scotia, The Baltimore Survey, The New Haven Study; all have been quoted to the point of becoming trite, but they do come out with some remarkably similar findings—about 20% of the population has a significant degree of handicap or disability due to mental, emotional or personality disorder, up to 60% have identifiable, diagnosable conditions, while up to 80% have some evidence of

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Approved for publication by the Commissioner, Department of Mental Hygiene and Hospitals.

personality or social maladjustment. The rates are much higher among the underprivileged, the poor, the displaced, the alienated, than among the affluent, the organized and the privileged, yet the poor get less and less adequate treatment. The needs are very great and are not being met. Traditional Psychiatry has long recognized this fact, but tended to shrug its shoulder and give the excuse that there were just not enough specialists to go around or enough man hours or that we had not yet developed sufficiently effective techniques or medications. We would just have to wait. In the meantime, the State Hospital remained the principle bastion of psychiatric care—for those who had developed full blown or chronic mental or emotional disorder, supplemented by private sanataria and general hospital units and public clinics and private offices—all overcrowded and many with waiting lists; still only dealing with a minority of the problems known to exist and demonstrated in repeated surveys.

Efforts have been made with some success to develop shorter methods of treatment and techniques such as family therapy and group therapy, behavior therapy, etc. The numbers of psychiatrists have doubled and redoubled in the past 20 years and other professionals have moved into the field giving therapy or counseling often after prolonged battles for a beachhead on the lucrative shores of private practice, or in clinics and hospitals won partly by default due to the shortage of psychiatrists. Even now most people with emotional problems are dealt with by non-mental health professionals or non-specialists, i.e., by general physicians, nurses, social workers, ministers, police and by lay advisors including bar tenders, cab drivers, etc., and this will continue to be true for the foreseeable future.

Confronted with these realities what can or should the mental health specialist do about it? We cannot any longer wait for the "breakthrough", the vague hope that some new "scientific discovery", a pill, a serum or whatever will bring any solution. It is becoming increasingly and overwhelming clear that mental, emotional, personality and behavior disorders have many causes and many effects. They cover the range of which human nature is capable and of the ills to which flesh is heir. Certainly heredity, biology, chemistry, metabolism, endocrinology and constitution are important, but also environment, family structure and functioning, accidents and illness, traumatic experiences, losses and deprivation, poverty and overcrowding, loneliness and isolation, fear and anxiety, alcohol and drugs. In other words, by the very nature of the disorders with which we are concerned and the complexity and variety of human nature and human experience it is unlikely that any single cause or single or simple cure for mental illness will ever be found. The same can probably be said for any category of mental disorders, such as the neuroses, psychoses, personality disorders or even of most of the specific illnesses or syndromes such as schizophrenia or depression or hysteria.

Is the situation then hopeless? If we do not know *the* cause or *the* cure, how can we treat, much less prevent, mental illness? Furthermore if there are not enough doctors or mental health professionals to treat the sick even if we knew how, or enough clinics, hospitals or other facilities in which to treat, even if we had the professionals to treat them or knew how to cure them, is not the situation discouraging to say the least? Not altogether. In the first place, before we panic we should remember that the world and its human population with essentially the same basic problems has been around for some time. Somehow we have managed to survive in spite of everything. In fact, currently we seem to be about to over-run the place, thanks to our successes. We have not only survived, but as William Faulkner so eloquently put it, we have prevailed—(so far). Mankind in common with the rest of nature, generally finds some way to cope. For the most part the best he can ever do by his own efforts is to assist nature

and perhaps undo some of his own mistakes, or relieve the consequences thereof.

What the physician tries to do is to help his patient cope a little better—to assist nature in healing or combating or preventing illness or disability in an individual patient. The Public Health Physician assumes or is delegated responsibility for the *health of a community*—a city or county or state. He is concerned about the incidence and prevalence of illness, about prevention or reducing the incidence and prevalence of illness. He is concerned about early case finding and getting cases to treatment before they become severe or chronic. He is concerned about rehabilitation to salvage and restore as much as possible of the functional capacity of those who have been ill or damaged. He is concerned about "positive health"—and such things as recreation, housing, community life and morale. The concerns of the Community Psychiatrist or Community Mental Health Specialist are identical with regard to Mental Health and Mental Illness in all the range of its manifestations. He also must work with and through and for the political authorities, the decision makers, the formal and informal power structure of the community—he must work with and through the established community institutions and agencies, the traditional caregivers who have already been helping or coping as best they can, but who might benefit from and usually welcome some expert assistance in the form of consultation about the mental health aspects of their patients, clients and activities. This might apply to public and private agencies or institutions, to medical and non-medical persons, to individuals and to organizations, to professionals and to laymen. He would be interested in working with and through already established resources, but also in spotting gaps in community resources and in promoting new ones to provide more adequate comprehensive coverage, more continuity of care, more attention to preventive aspects or rehabilitation.

Community psychiatry is a relatively new subspecialty or development within the field of psychiatry although many psychiatrists have long given some time and attention to community efforts. Certain other developing areas within psychiatry have been closely analogous and have made contributions to theory and practice in community psychiatry—such as Industrial Psychiatry, School and College Psychiatry and Military Psychiatry.

Yes, there are things that can be done, even with our present knowledge, but obviously there is urgent need for research, basic and applied, for trial and evaluation, for pilot projects in new methods of more effective treatment and prevention—for bringing to bear the insights and skills of other professions and disciplines—the law, the ministry, education, business administration, the social sciences, etc. There is the need for better, more imaginative utilization of other personnel, professional, semi-professional and lay volunteers. Obviously, community psychiatry is no panacea or millennium. Neither is it a substitute for traditional methods and approaches.

Community psychiatry has not been without its detractors and resistance. It has been called the "latest band wagon", "political psychiatry", "socialized psychiatry", etc. Some psychiatrists ask "Is this psychiatry at all?" and others ask "What's new about it? We've been doing that all along." It also has had its drum beaters and is in danger of being oversold and of making unrealistic and certainly unproven claims. To me it is supplementary and complementary. It seeks to be comprehensive—to integrate, coordinate, organize, implement, facilitate, support and promote all forces, efforts, knowledge and experience in the direction of improving mental health of all the population of a community.

What, you may ask, is "the mental health of a community"? Well, it is at least the minimum of mental illness and disability. It is the lowest possible incidence of illness, hospitalization, divorce, crime, delinquency,

absenteeism, school dropouts, drug addiction and alcoholism, abandoned, neglected and mistreated children. It is the highest possible level of employment, productivity, creativity, morale, health and well being, yes, even the greatest pride, happiness, joy and satisfaction in living. It is the minimum of boredom, loneliness, and frustration, fear, anxiety, insecurity, anger, hostility, and suspiciousness. One may say this is utopian, unrealistic fantasy, but I submit that these are real, practical and very important goals that make a very real difference in determining rates of mental illness and levels of mental health, also of physical illness and physical health.

This is obviously not just the job or concern of psychiatry, but of every segment of medicine, of the community—of every person of good will. Mental health professionals and sub-professionals can make a significant contribution if we raise our sights, broaden our scope, come out from behind our desks, our walls, our narrow vision, our restricted traditions, our class prejudices and learn to work together. This will not bring the millennium, but it will be progress.

We will have to be concerned with poverty, housing, working conditions, education, racial tensions, law enforcement practice and problems, city planning, and the total health needs of all sections and segments of the population, not just with the case who comes or is sent to us for treatment. We will have to utilize and develop new kinds of professionals who can help in this task. We will have to develop consultation as a major skill and method of operating for mental health experts. This is in response to community need and demand —not something imposed by government or by the profession. In fact, the community and the non-professionals are more often ready to accept change than are professionals who have a vested interest in the status quo. These trends, developments, demands and needs also concern medical schools and training centers and even colleges and high schools where the training and preparation of new varieties of health personnel will be developing. Certainly the training of psychiatrists and clinical psychologists will have to include more community orientation.

The Community Mental Health Center is an important aspect of community psychiatry and it incorporates many basic principles, but it is not synonymous. It is a new and more comprehensive type of mental health facility to help serve the needs of a community. Obviously all psychiatrists will not and should not do community psychiatry as such. There will always be a need for private office psychiatry, hospital psychiatry, academic psychiatry, etc. I predict that more psychiatrists in the future will be more extensively involved in the community—in doing consultations to community agencies, etc., and that some will move into specialization in this field. This is a relatively new, rapidly evolving area, the boundaries of which are constantly changing. There is the urgent need for basic and clinical or operational research both in new and more effective techniques in delivering service and in methods of prevention.

These new and better methods of supplying service and meeting needs will obviously be more expensive and will require more personnel and more facilities. There is no pretense that these programs will save money or will be provided by a beneficent government, federal or state, although both probably will and should participate. If the public and the communities really want better mental health programs, treatment, care, rehabilitation, and prevention, they will have to accept a share of responsibility and cost just as they have for physical public health. Good community mental health may be expensive but at least it aims to get results, to avoid throwing good money after bad. It refuses to accept the proposition that all society can do is "take care of" the mentally ill for the rest of their lives in human warehouses, which is possibly more expensive in the long run.

The Medical Society of Virginia . . . .

National Conference for Nurses and Physicians

What to do! and How to do it! was the theme of the Fourth National Conference for Nurses and Physicians held on October 6, 1967 at, AMA Headquarters in Chicago.

The conference was aimed at strengthening relationship between the medical and nursing professions on the state level; getting a comprehensive view of what has and is being done in the area of physician-nurse relationships and planning state-wide conferences based on the National AMA-ANA Conferences.

Dr. Eleanor C. Lambertson, director of the Division of Nursing Education, Teachers College, Columbia University, discussed "Current Nursing Education and Practice". She pointed out that revolutionary changes are being made in nursing education which are to correspond to social changes, dynamics, and greater demand for expansion of health services. The various specialties and subspecialties require specially trained nursing personnel. Heart surgery, pediatrics, and psychiatry are examples. She "envisioned" three levels of nursing: clinical practitioner, general practitioner and supportive nursing personnel.

Sister M. Raphael, R.N., M.S., C.S.C., Director of the School of Nursing, Holy Cross Hospital, Salt Lake City, Utah, and Joseph W. Marshall, M.D., of Twin Fall Hospital, Idaho, discussed at length the "Development and Activities at a Joint Physician-Nurse Liaison Committee in the State of Idaho", their final result was the establishing of a joint conference committee of physicians and nurses. It is my opinion that Virginia has an adequate and commendable Liaison Committee between the two professions. However, I seriously doubt if the deliberations of our joint committee meetings and the standards outlined by the two professions have been adequately distributed among either profession.

Discussions were quite enlightening. Universally, there is a critical shortage of nurses everywhere. Diploma nurses now constitute 80% of the nursing profession. Baccalaureate Degree and Associate Degree nurses will eventually replace diploma nurses. In New York State no more diploma or LPN schools can be instituted after January 1, 1967. During the past eight years the total number of Diploma Schools have decreased from 918 to 797. New Hampshire State subsidizes Diploma Nursing Schools. Baccalaureate Degree and Associate Degree nurses will require in-hospital orientation similar to internship and residences for physicians before becoming competent nurses.

I sincerely trust that the medical and nursing professions in the State of Virginia will work more closely together to develop a better understanding of each others problems, establish better relationship in securing the best and most efficient patient care obtainable.

I appreciate having the honor of representing The Medical Society of Virginia at this meeting.

W. N. Thompson, M.D., *Chairman*
Liaison Committee with Nurse Examiners and Organized Nursing

Woman's Auxiliary . . . .

Treats in Fun Raising are abundant in several of the local auxiliaries these days. The Woman's Auxiliary to the Richmond Academy of Medicine is holding its Sixth Annual Antique Show and Sale on the twelfth of March. The proceeds from the show are earmarked for the benefit of the Crippled Children's Hospital, Sheltering Arms Hospital and for a Grant-In Aid program to assist medical students at the Medical College of Virginia. It is interesting to note the need in this area —over one-third of the medical students find it necessary to seek some kind of outside financial assistance. . . . Meanwhile, the Auxiliary to the Medical Society of Virginia Beach is busy selling tickets to a theater party—the Little Theater's production of "The Late Christopher Bean". The story of a doctor no less! The proceeds from the show will go towards the Arts and Crafts Room which the Auxiliary maintains at the Virginia Beach Hospital. . . . Kay Reardon writes from Fairfax that they are busy getting plans under way for a horse show next Fall to raise funds for A.M.A.-E.R.F. . . . And Nancy Baker, President of the Woman's Auxiliary to the Alexandria Medical Society writes that they are selling candy for their A.M.A.-E.R.F. Fund. They also held a Coppercraft Show and won for the Auxiliary a chafing dish which they are selling chances on. These funds go towards their Student Nursing Scholarship and the Day Room of the Psychiatric Unit at the Alexandria Hospital, which they have recently furnished.

ANNOUNCING—The Mid-Winter Board Meeting—will be held at The Medical Society of Virginia headquarters, 4205 Dover Rd., Richmond, on Thursday, March 28th. The meeting will be called to order at ten o'clock. Mrs. Anderson has requested that in lieu of roll call, she would like for each President of a county auxiliary to give a one to two minute report on their most outstanding "Treat in Service to Others" —either completed or planned for this Auxiliary year. The Board Meeting will be followed by a Workshop and luncheon—featuring VAMPAC and AMPAC, as represented by Mrs. Ethel Gastineau, who is the only woman member of the AMPAC Board. It really is a privilege to hear this charming speaker—who is guaranteed to make us all realize how very vital AMPAC is. We will have "The Package Deal—Continued" and in addition to AMPAC and VAMPAC, there will be the A.M.A.-E.R.F. Package, the Community Health Package and a film and report on "Meals On Wheels". The Richmond Auxiliary will provide the luncheon for the benefit of A.M.A.-E.R.F. The President is particularly eager to know who will be attending the A.M.A. Meeting in San Francisco on June 16th through 20th. We will want our full delegation from Virginia of five members plus our President. Be certain to get in touch with Mrs. Daniel Anderson if you plan to go.

For "A Treat in Service to Others" remember to collect your sample drugs. The Woman's Auxiliary to the American Medical Association recommends that the following guidelines be observed in the collection of drug samples from physicians:

1. Sample drugs should be collected from the physician's office in their original unopened packages only by members of the Woman's Auxiliary. These samples should be put in a carton, sealed, and sent to a responsible collection agency. Drugs collected by the Auxiliary shall not be sent directly overseas.

2. The responsible collection agency (registered with the U.S. Food and Drug Administration) will, under the supervision of a registered pharmacist at that agency's place of business, sort and screen all samples to eliminate all recalled, out-dated and investigational drugs.

3. Sample drugs, after having been screened and sorted, will be sent in the original, unopened package, by the agency to physicians and hospitals overseas.

Mrs. Joseph M. Straughan, Box 856, Wise, Virginia, is the State International Health Chairman, and will be more than happy to help you get an active program for the collection and disposition of drug samples underway.

A New Opportunity for "A Treat in Service to Others"—is the A.M.A. Blood Program. At the 24th Annual Conference of State Presidents, Presidents-elect, National Officers and Chairmen, Mrs. Herbert J. Ulrich, Chairman of Community Health for the Auxiliary, based her entire talk on the A.M.A. Blood Program. The following are excerpts from her speech: "We find that at present one out of every 80 persons in the United States receives a transfusion each year, and one out of every 20 transfusions saves a human life. Since the human body is the only source of blood, and since only about one-half of the total population is able to donate, the solution to the blood shortage problem is to get some of the hundred million non-donating potential donors to donate, as well as getting present donors to donate more frequently . . . Since blood cannot be produced artificially and can only be obtained from a human being, and since different types of blood must be available on short notice, problems are manifold. Stockpiling of blood as a means of buffering the random fluctuations in both supply and demand is impossible, since the lifetime of stored blood is limited to only 21 days. Therefore, donors must be of prime concern. Many recruitment methods have been used, such as: advertising and personal contact; offers to pay donors; reaching the past donors; "Replacement fee" to encourage donations from recipients of blood as well as from their families and friends; those participating in blood insurance programs; and lists of "rare" donors. However, despite all the recruitment methods, now that you've become aware of this alarming shortage, notice how many times a week an item appears in your local newspaper pleading with the public to come to the fore and donate blood. Can we with our #1 Objective "To assist the A.M.A. in its program for the advancement of medicine and public health" ignore this grave situation?" Mrs. Ulrich suggests that we, as auxiliary members, develop public informational programs, offer to assist in local voluntary donor programs, strengthen and support existing programs, and that we, our families and friends participate by being regular donors.

Editorial . . . .

Progress in Obstetrics

THE AVERAGE twenty-five year period is not long in the history of medicine. However, the past quarter century, like the same interval before it, has seen great changes in all branches of medicine. The greatest notice has been given to advances in surgery and internal medicine. Obstetrics has also changed. Recognition of the fact that some of our problems of yesterday have been solved may make more endurable those which remain with us.

Some problems seem less pressing rather than resolved. Eclampsia has become a rare condition in private practice today. Pre-eclampsia is also far less evident than it was at the beginning of the careers of many of us. Why this should be true is difficult to explain. Hard-headed patients still persist in gaining too much weight. Diuretics and anti-hypertensives would seem to have had only a minor preventative role in this change. They have reduced somewhat the terrors of the disease when used as treatment. Some wit might associate the decline of toxemia with the rise of the hem-line.

Likewise, in our own private practice, we see now far fewer severe cases of premature separation of the placenta. In the not-so-distant past we used to see several cases a year of patients in shock due to this condition. Perhaps it is mere luck or coincidence that none has come our way in more than three years. It is true that we have prescribed folic acid liberally, but recent studies have spread some doubt as to whether folinate begun after the first months does any good.

The rare case of hypofibrinogenemia, a condition which occasionally is associated with premature separation, and with missed abortion, is now fairly simply treated with fibrinogen. The greater sophistication and availability of banked blood have largely eliminated the other sources of maternal mortality due to hemorrhage in the ante- and post-partal periods. Another factor in the prevention of postpartum hemorrhage is the fact that we no longer hesitate to explore the postpartal uterus immediately in the search for retained secundines. Some of us do it routinely.

Making us braver in the last procedure is the knowledge that we have many antibiotics at our command. Even so, antibiotics actually seem less needed now that we have them than in the days of our residencies when concern for postpartal infection was daily in our minds.

The availability of antibiotics as well as of banked blood probably account for an apparent nation-wide slow relaxation of the indications for Caesarian section. A recent author seems to feel that nearly a fifth of all patients presenting themselves in labor with the breech presenting should be sectioned.

Uterine inertia is still with us. Even today it is the indication for an occasional section. However, one of the greatest boons to all concerned with dilatory labors, particularly the first, has been the introduction of the use of slowly administered intravenous pitocin. Certainly this development has been a great step in preventing the need for instrumental interference as well as section. Now the obstetrician tempers his patient with pit.

The recognition of the cause of erythroblastosis has had perhaps more popular magazine appeal than any other of our advances in this period. Treatment of the afflicted babies with exchange transfusion has saved many. But prevention of the trouble is of more interest. Several ideas have been proposed and set aside. The newest hope comes from the possible development of a sort of immunization of the mother. Intrauterine transfusion of the probably afflicted child has been tried with some success. The need for this procedure is so uncommon that perhaps candidates for it should be sent to large centers where more experience can be attained.

Most recent developments seem to be refinements of old techniques or new ways of handling rare problems. Some of these latter are of questionable safety. The proper way of treating intrauterine fetal death has long been a worry. Usually such cases are resolved spontaneously. There has been some interest in and success with the intrauterine injection of 20% saline or of hypertonic glucose as a means of promoting the expulsion of such undesirable uterine contents. Shock and death have occurred with the first technique and septic death with the second.

There are still many puzzles. We do not yet know the cause of eclampsia, of premature separation, or of premature rupture of the membranes. Prematurity itself is a constant problem.

Much basic research is being done and this may one day supply the answers to these and to other problems. Genetic research and practice are advancing. The chromosomal geography of some defects is being plotted. Many parents of defective children can now obtain advice as to the dangers of future child-bearing. Genetic planning is in its infancy. It is possible that future generations will see genetic manipulations. This may prove to be good or evil, but the thought of government control of such a personal process and right as reproduction is rather chilling.

Considerable research is being done upon placental physiology. The former idea that the placenta functions as a sort of arterio-venous shunt is being questioned. Blood, and thus the nutrients, may be supplied in spurts and unevenly. Is this significant? The placenta seems to be selective in its reception of offered substances. Some large molecules and some smaller ones do not cross the placental "barrier" while others do. Insulin, for instance, is prohibited. Is the rejection of the fetus and placenta at labor similar to the rejection of a homograph? Is, then, premature separation the result of some sort of allergic reaction? Placental research is very difficult but it may one day supply the answers to the problems of prematurity, defective infants, premature separation, and toxemia. Once the facts are known some way of encouraging the normal development of the placenta may be found.

GORDON W. JONES, M.D.

Medical Note

WITH THE UNITED STATES in an ever-expanding war with nearly 20,000 Americans dead and over 100,000 wounded, and with casualties steadily increasing so that newspapers now list only the names of those killed from the immediate area, it is heartening to know that our leaders are fully aware of how widespread human suffering is in our land and how deftly they can place it all in the proper perspective.

These thoughts were prompted by the following Associated Press release from Washington which appeared the same day the Viet Cong overran South Vietnam.

> "President Johnson, while meeting Tuesday with March of Dimes poster child Timmy Faas, disclosed that his 7-month-old grandson burned his finger when he dipped it in a bowl of chili. The incident occurred last Saturday at the LBJ Ranch in Texas. A White House spokesman said Patrick Lyndon Nugent stuck his hand in a bowl of hot chili and was treated by a doctor for slight finger burns. He is 'fine now' the spokesman added."

H. J. W.



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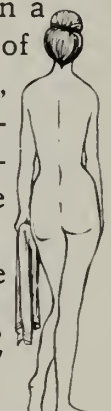
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News . . . .

Calendar of Events

Clinical Cardiology—19th Annual Post-Graduate Day Program of Roanoke Memorial Hospital—Roanoke—March 21-22, 1968.

Second National Congress on Socio-Economics of Health Care—The Palmer House—Chicago, Illinois—March 22-23, 1968.

Southeastern Surgical Congress—Annual Meeting—Sheraton Park Hotel—Washington, D. C.—March 25-28, 1968.

Newer Aspects of Cardiac Therapy—Symposium presented by Roanoke Valley Heart Association—Hotel Roanoke—Roanoke—2:00 p.m.-5:00 p.m., March 28, 1968.

21st National Conference on Rural Health—Olympic Hotel—Seattle, Washington—March 29-30, 1968.

Second National Congress on Medical Ethics—Drake Hotel—Chicago, Illinois—March 30-31, 1968.

American College of Physicians—49th Annual Session—In association with the Royal College of Physicians of London—Sheraton-Boston Hotel—Boston, Massachusetts—April 1-5, 1968.

Annual Clinical Conference—Louise Obici Memorial Hospital—Suffolk—April 3, 1968.

Annual Joint Cardiac Symposium—Sponsored by Heart Association of Northern Virginia and Washington Heart Association—Marriott Twin Bridges Motor Hotel—Arlington—April 10, 1968.

Cardiovascular Responses to Anesthesia—Fifth Annual Spring Symposium of Virginia Society of Anesthesiologists—Sheraton Motor Inn—Richmond—April 19-21, 1968.

Virginia Society of Opthalmology and Otolaryngology—49th Annual meeting—The Cavalier—Virginia Beach—May 2-4, 1968.

The Child With Heart Disease—Recognition and Management—Continuing Education Program of the Medical College of Virginia—Richmond—May 9-10, 1968.

Virginia Academy of General Practice—Annual Scientific Assembly—The Chamberlin—Fort Monroe—May 9-12, 1968.

Medical College of Virginia Alumni Scientific Assembly—Richmond—May 31, 1968.

Seaboard Medical Association—Annual Meeting—Nags Head, North Carolina—June 21-23, 1968.

9th Annual Cardiovascular Symposium—Sponsored by Tidewater Heart Association and Council on Clinical Cardiology, American Heart Association—Lake Wright Motel, Virginia Beach—September 6-7, 1968.

New Members.

The following members were received into The Medical Society of Virginia during the month of January:

James Porter Baker, M.D., Richmond

Henry Simon Campbell, M.D., Martinsville

William Hardy Chapman, M.D., Charlottesville

Thomas Wade de Beck, M.D., Charlottesville

Larry Richard Denius, M.D., Hot Springs
Jack Merrit Gwaltney, Jr., M.D., Charlottesville
Juan Marcelino Gonzalez, M.D., Farmville
John Norman King, M.D., Norfolk
John Wilson Kolmer, M.D., Roanoke
Edmund Dillahunty Lowney, M.D., Richmond
Bernard Allan Lublin, M.D., Richmond
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John Charles Rhoads, M.D., Manassas
William Sproul Stickley, M.D., Winchester
Lawrence Elwood Valentine, M.D., Accomac
Carl Vernon Yutzy, M.D., Newport News

Local Committee on Arrangements.

The committee on arrangements for the annual meeting of The Medical Society of Virginia in Roanoke, October 13-16, is already at work and, as usual, an excellent meeting can be expected. The committee is as follows: General Chairman, Dr. Robert L. A. Keeley; Scientific Exhibits, Dr. Reuben D. Knopf; Technical Exhibits, Dr. John Cole, Jr.; Hotels and Halls, Dr. Thomas E. Strong; Entertainment, Drs. Robert S. Creekmore, Max E. Bertholf, Frank M. Guilfoyle, and J. Milton Miller; Press, Dr. William S. Erwin, Jr., and Golf, Dr. Philip C. Kistler.

Albemarle County Medical Society.

Dr. Armistead Page Booker has been elected president of this Society, with Dr. William B. Pollard, vice president and president-elect, and Dr. Charles H. Gleason as secretary-treasurer. All officers are from Charlottesville.

Drs. Stoneburner and Gearing Honored.

Shenandoah County Memorial Hospital staff members recently paid tribute to their two senior physicians, Dr. Ralph W. Stoneburner, of Edinburg, and Dr. F. Wilson Gearing, of Woodstock. The occasion was in honor of their birthdays—Dr. Stoneburner was 80 on January 13th, and Dr. Gearing was 76 on January 16th. Between them they have practiced 101 years, almost all of it in Shenandoah County. Both doctors were making their daily rounds of hospital patients when they were summoned to an "important conference" which turned out to be a surprise party.

Dr. Frank M. Johnson,

Roanoke, has been elected lieutenant governor of Division 2 of the Capital District, Kiwanis International. He is a member and former president of the Williamson Road Kiwanis Club.

Hospital Staff Members.

Dr. J. M. Straughan has been elected chief of staff at St. Mary's Hospital in Norton. Dr. D. B. Jones is secretary; Dr. J. T. Phillips, Jr., chief of surgery; Dr. U. S. Gonzalez, chief of medicine; Dr. Lewis K. Ingram, chief of obstetrics and gynecology; and Dr. Don S. Park, chief of pediatrics.

The Annual Clinical Conference

Of the Louise Obici Memorial Hospital, Suffolk, will be held on April 3rd. The subject for discussion is Problems in the Management of Breast and Lung Cancer, and speakers will be: Dr. Felix Wroblewski, Brooklyn, New York; Dr. Walter Lawrence, Richmond; Dr. Walter Gobbel, Nashville, Tennessee; Dr. Norman Delarue, Toronto, Canada; Dr. John Evans, Durham, North Carolina; and Dr. Melvin Samuels, Houston, Texas.

The luncheon speaker will be Dr. William Endicott of the National Cancer Institute.

The annual award for the outstanding contribution to medicine will be presented to Dr. Kinloch Nelson, Dean of Medicine of the Medical College of Virginia.

Medical and Chirurgical Faculty of Maryland.

The annual meeting of the Faculty will be held April 17-19, at the Alcazar, Baltimore. Speakers include Dr. J. Hartwell Harrison, Peter Bent Brigham Hospital, on The Surgical Management of Disorders of the Adrenal Cortex, Dr. Charles M. Caravati, Medical College of Virginia, on Cirrhosis of the Liver; Dr. F. Mason Sones, Jr., Cleveland Clinic Foundation, on Cine Coronary Arteriography in Evaluating Medical and Surgical Therapy for Coronary Atherosclerosis; Dr. Maurice B. Strauss, Tufts, University, on Chronic Nephritis Revisited; Dr. Arthur M. Vineberg, The Royal Victoria Hospital, on Cardiac Surgery; Dr. Clifford F. Gastineau, Mayo Clinic, on Care of the Unstable Diabetic; Dr. Louis M. Hellman, State University of New York, on Safety of Oral Contraceptives; and Dr. Stanley Baum, University of Pennsylvania, on Recent Advances in X-Ray Diagnosis of Surgical Diseases. There will also be Medical and Surgical Grant Rounds presented by Johns Hopkins University School of Medicine and the University of Maryland School of Medicine.

Following the Baltimore meeting, there will be a Caribbean Cruise Convention from April 21 to 28, during which there will be daily scientific lectures and films.

All physicians are invited to attend the above sessions and the cruise and a detailed program may be obtained from the Faculty at 1211 Cathedral Street, Baltimore, Maryland 21201.

Medical Symposium.

The 2nd Annual Continuing Education Medical Symposium was held on February 23-24, by the Medical Staff of the Norfolk General Hospital. Norfolk speakers were Dr. Howard I. Kruger, Case Report of Cryptococcosis; Dr. Clarence B. Trower on Chronic Cystic Mastitis; Dr. Claude A. Smith, Case Report of Feto-Maternal Transfusion; and Dr. David B. Kruger, Case Report of Conversion Reaction. Guest speakers were Dr. George S. Richardson, Pittsburgh, on Restoration of Function in Hand Injury; Dr. Bentley P. Colcock, Boston, on Inflammatory Lesions of the Colon; Dr. John R. Ross, Boston, on The Gastrointestinal Biopsy Capsule as a Diagnostic Tool; Dr. Robert E. Wise, Boston, on Current Concepts of Cholangiography; Dr. Kenneth Savard, Miami, on Recent Findings in the Endocrinology of the Human Ovary; and Dr. George F. Bond, Department of Navy, on Medical Aspects of Undersea Living.

Wanted.

Qualified anesthesiologist to head department at C. & O. Hospital, Clifton Forge, Virginia. Income in excess of $30,000, largely on fee for service basis. Apply to Dr. J. M. Emmett, C. & O. Hospital, Clifton Forge, Virginia 24422. (*Adv.*)

Hospital Physicians Wanted.

The Medical Staff, Board of Directors, and administration of Winchester Memorial Hospital, Winchester, are agreed to a plan of four full time physicians to be in private general practice limited strictly to the Emergency Department of the hospital. Average work week is forty-two hours. A generous guarantee has been established.

Four experienced physicians having a Virginia license are needed. The target date is July 1, 1968. Interested physicians contact the committee chairman, Dr. James A. Miller, Box 554, Winchester, Virginia. Telephone 662-0377 area code (703). (*Adv.*)

Location Wanted.

Anesthesiologist, FACA, wants relocation to State of Virginia. Tidewater area preferred. Licensed, experience, fee for

service preferred, group or partnership accepted. Write #135, care Virginia Medical Monthly, 4205 Dover Road, Richmond, Virginia 23221. (*Adv.*)

Obituary . . . .

Dr. Charles Marsden Irvin,

Roanoke, died January 4, following a heart attack. He was seventy-five years of age and graduated from the Medical College of Virginia in 1921. Dr. Irvin had served as coroner and medical examiner in Roanoke for forty-four years and was city health commissioner for twelve years. He had been a member of The Medical Society of Virginia for forty-five years.

The following resolutions were adopted by the Roanoke Academy of Medicine, at its meeting on January 8:

Dr. Charles Marsden Irvin beloved and respected member of The Roanoke Academy of Medicine died January 4, 1968, in a local hospital following a heart attack.

Dr. Irvin was born in Roanoke, June 12, 1892. He was an alumnus of Roanoke College and received his M. D. degree from the Medical College of Virginia. He served as house surgeon at St. Lukes Hospital under Dr. Stuart McGuire and was resident surgeon for one year at the City Hospital in Richmond.

He was a veteran of World War I and practiced medicine in Roanoke since 1922. He served as Coroner and Medical Examiner for the city for forty-four years and was appointed City Health Commissioner in July 1954 and held that post until 1956. He also served on the State Board of Medical Examiners and for the past ten years was medical advisor to the Blue Cross and Blue Shield.

He was a member of the American Medical Association, The Medical Society of Virginia and past president of The Roanoke Academy of Medicine. He was a member of The First Presbyterian Church, the Elks Lodge and throughout his life enjoyed hunting and fishing.

Dr. Irvin is survived by his wife, a daughter, a brother and five sisters, most of whom reside in Roanoke.

We join his family, the community, his many friends and patients in mourning his death and

Be It Resolved that The Roanoke Academy of Medicine enter in its minutes these remembrances and that copies be sent the family and the Virginia Medical Monthly.

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Henry Lee, M.D.
Charles A. Hefner, M.D.



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Turn page for contraindicati[illegible]recautions and side effects.

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The therapeutic action is smooth, and persits for a full 24 hours. With Enduron you can prescribe convenient once-a-day dosage without skimping your patients on day-long thiazide effectiveness.

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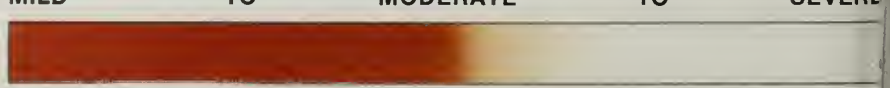
Use Enduron as a basic therapy in patients with mild to moderate hypertension. A single 5-mg. tablet each day is ample in most cases.

Once a day, every day

ENDURON

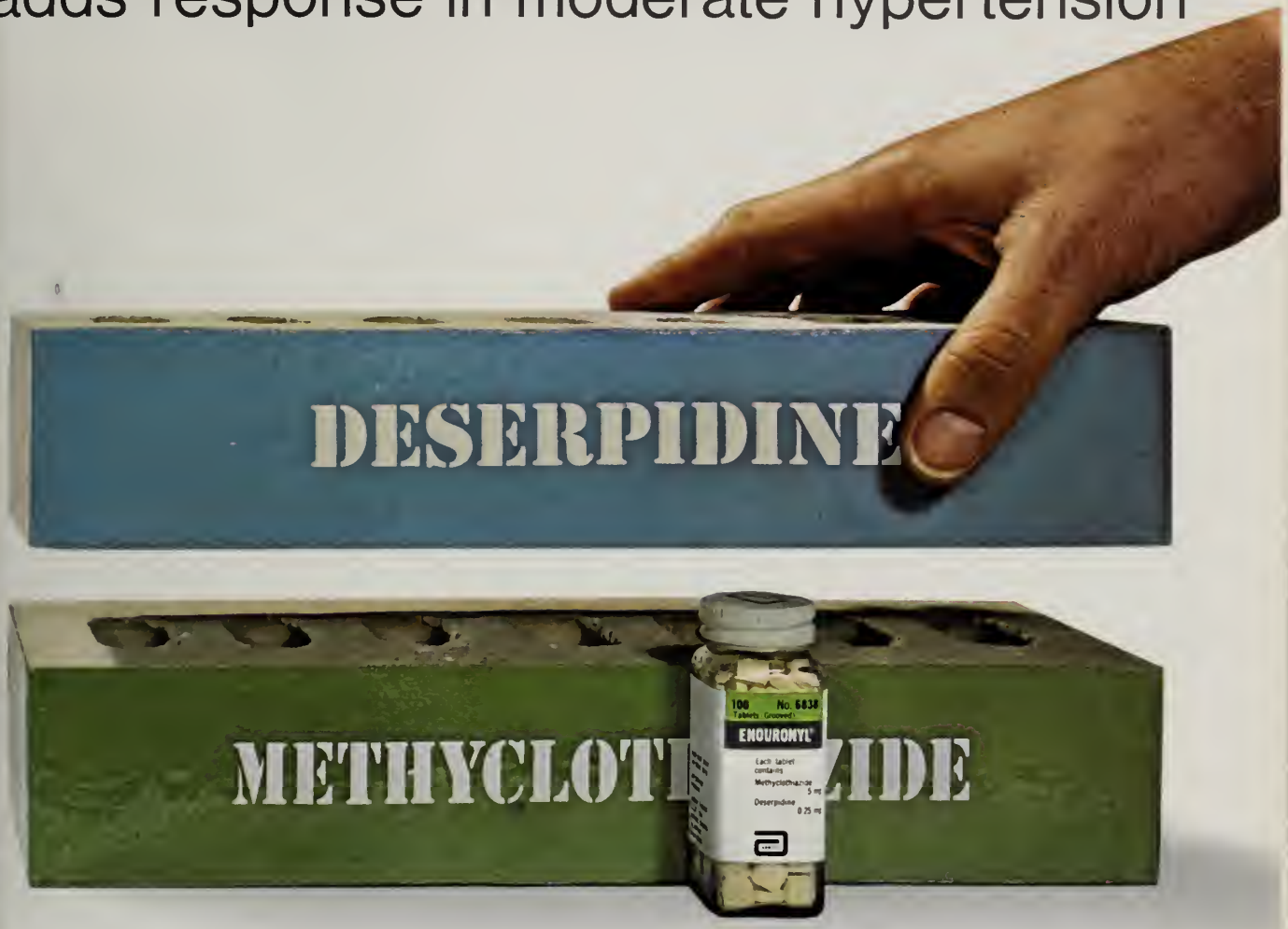
METHYCLOTHIAZIDE

MILD TO MODERATE TO SEVERE



See Brief Summary on final page of advertisement

Enduronyl: Its deserpidine component adds response in moderate hypertension



Less frequent rauwolfia side effects than with reserpine

When you wish to build further response, consider shifting to Enduronyl.

Enduronyl adds a building block of *deserpidine*. This is a purified rauwolfia alkaloid available only from Abbott. It adds good antihypertensive and tranquilizing activity. Yet its incidence of untoward effects, particularly lethargy and depression, is lower than with reserpine.

Enduronyl is available plain or Forte. The latter provides its variation where most helpful, by doubling the deserpidine.

Use Enduronyl for patients throughout the broad range of mild to moderately severe hypertension.

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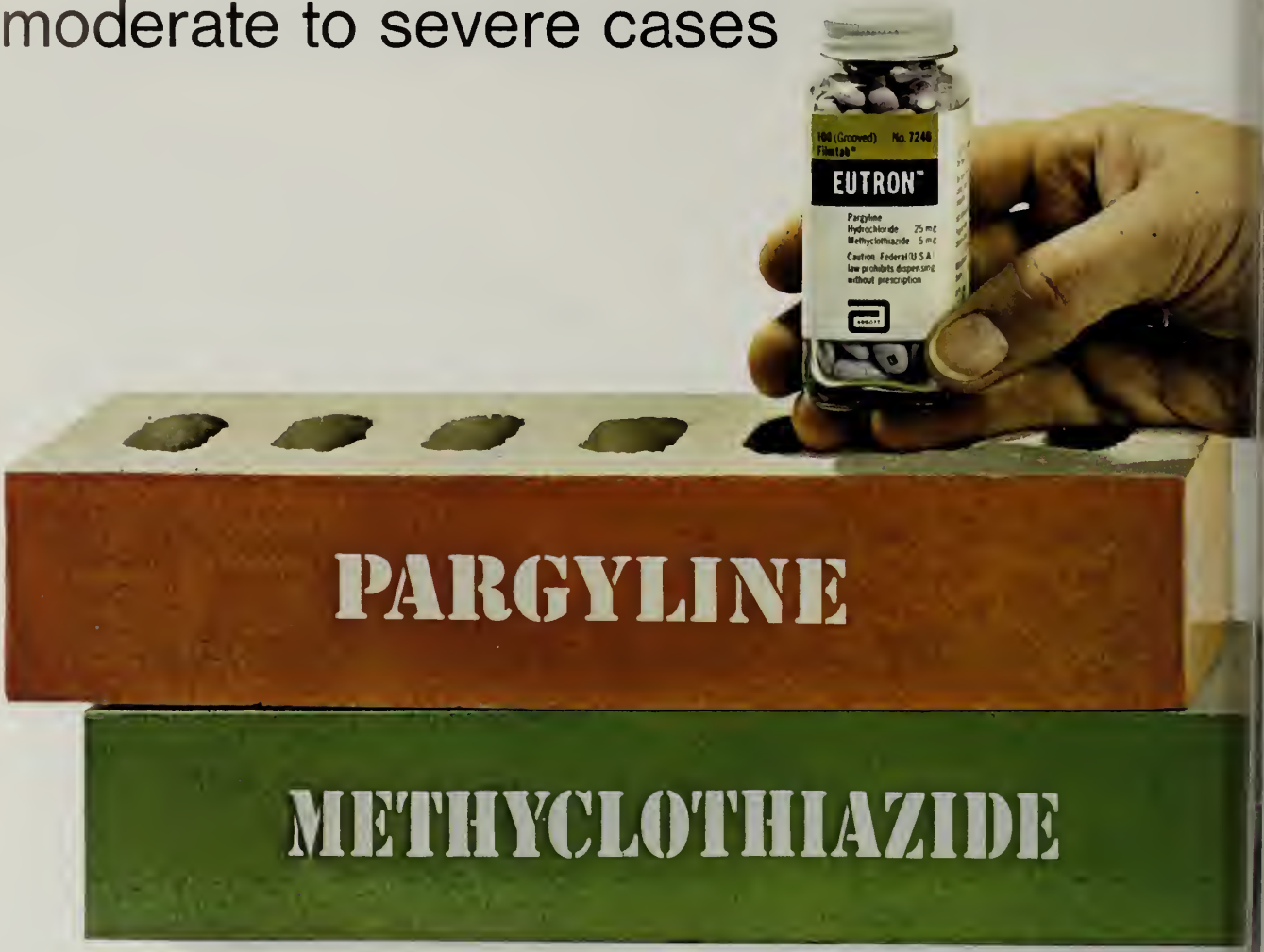
METHYCLOTHIAZIDE 5 mg. with DESERPIDINE 0.25 mg. or (FORTE) 0.5 mg.

MILD TO MODERATE TO SEVERE



See Brief Summary on final page of advertisement

Eutron: A unique combination for handling moderate to severe cases



Affords almost uniform diastolic reduction in all body positions

Eutron lowers diastolic pressures nearly equally, whether your patient is standing up or lying down.

Thus, in clinical trials, average *standing* diastolic readings were reduced from 112 pre-treatment to 90 post-treatment; *sitting* from 115 to 95; and *recumbent* from 112 to 94.

Note that following Eutron, the diastolic reductions were nearly alike in all three body positions.

Use Eutron for managing your moderate to severe cases. Its building blocks enhance each other; hence lesser doses often suffice.

Once a day, every day

EUTRON™

PARGYLINE HYDROCHLORIDE 25 mg.
with METHYCLOTHIAZIDE 5 mg.

MILD TO MODERATE TO SEVERE



See Brief Summary on final page of advertisement

801094

ENDURON® | ENDURONYL®

METHYCLOTHIAZIDE

Each tablet contains
Methyclothiazide 5 mg. with
Deserpidine 0.25 mg. or 0.5 mg

Indications: Edema and mild to moderate hypertension (Enduron), and mild to moderately severe hypertension (Enduronyl). More potent agents, if added, can be given at reduced dosage.

Contraindications: Sensitivity to thiazides; severe renal disease (except nephrosis) or shutdown; severe hepatic disease or impending hepatic coma (hepatic coma due to hypokalemia has been reported in patients on thiazides). Do not use Enduronyl in severe mental depression, suicidal tendencies, active peptic ulcer, or ulcerative colitis.

Warnings: Consider possible sensitivity where there is history of allergy or asthma. If added potassium is indicated, dietary supplementation is recommended. Reserve enteric-coated potassium tablets for cautious use only when necessary, as they may induce serious or fatal small bowel lesions (stenosis with or without ulceration), cause obstruction, hemorrhage, and perforation often requiring surgery; discontinue them immediately if abdominal pain, distention, nausea, vomiting, or g.i. bleeding occurs. Neither Enduron nor Enduronyl contains added potassium.

Precautions: Use thiazides cautiously in severe renal dysfunction, impaired hepatic function or progressive liver disease; also in pregnancy (bone marrow depression, thrombocytopenia, and altered carbohydrate metabolism have been reported in certain newborn). In surgery, thiazides may reduce response to vasopressors, and increase response to tubocurarine. Antihypertensive response may be enhanced following sympathectomy. Watch for electrolyte imbalance (e.g., hyponatremia) in all patients. In hypokalemia (especially in digitalized patients) give supplemental potassium. In hypochloremic alkalosis, give supplemental chloride.

Use rauwolfias with caution in patients with history of peptic ulcer. Rauwolfias with anesthetics may produce hypotension and bradycardia. Discontinue Enduronyl two weeks before elective surgery. Consider vagal blocking agents during emergency surgery. In epilepsy, adjust anticonvulsant dosage. In electroshock, shorten stimulus strength and duration. In occasional patients with depressive tendencies, rauwolfias may precipitate severe mental depression that usually disappears when drug is stopped.

Adverse Reactions: Thiazide reaction include blood dyscrasias (thrombocytopenia with purpura, agranulocytosis, aplastic anemia); elevation of BUN, serum uric acid or blood sugar; anorexia, nausea, vomiting, diarrhea, headache, dizziness, paresthesia, weakness, skin rash, photosensitivity, jaundice, symtomatic gout, and pancreatitis. Cutaneous vasculitis in the elderly has been reported with other thiazides. Adverse effects with deserpidine are qualitatively similar to those with reserpine, but their incidence is lower. These include nasal stuffiness, abdominal cramps or diarrhea, nausea, headache, weight gain, reduced libido and potency, peptic ulcer aggravation, epistaxis, skin eruption, asthma in susceptible patients, electrolyte imbalance, excessive salivation, and a reversible Parkinson's syndrome. Excessive drowsiness, fatigue, weakness, and nightmares may signal mental depression. Thrombocytopenia, purpura, and a symptom manifested by dull sensorium, deafness, uveitis, glaucoma, and optic atrophy are rare allergic reactions to other rauwolfias. Hypotension from antihypertensive agents may precipitate angina attacks in susceptible individuals. Usually adverse reactions disappear when drug is withdrawn.

EUTRON™

Each tablet contains
Pargyline Hydrochloride 25 mg.
with Methyclothiazide 5 mg.

Indications: For treatment of patients with moderate to severe hypertension, especially those with severe diastolic hypertension. Not recommended for patients with mild or labile hypertension amendable to therapy with sedatives and/or thiazide diuretics alone. It is desirable to establish the dosage of Eutron by administering component drugs separately.

TM-TRADEMARK

Contraindications: Pheochromocytoma, advanced renal disease, increasing renal dysfunction, paranoid schizophrenia and hyperthyroidism. Hepatic coma has been reported as consequence of hypokalemia with thiazide therapy. Until further experience is gained not recommended for patients with malignant hypertension, children under 12, or pregnant patients.

Concomitant use of the following is contraindicated: other monoamine oxidase inhibitors; parenteral forms of reserpine or guanethidine; sympathomimetic drugs; foods high in tyramine such as cheese; imipramine and amitriptyline, or similar antidepressants; methyldopa. 2 week interval should separate therapy and use of these agents.

Methyclothiazide is contraindicated in patients with known sensitivity to thiazides.

Warnings: Pargyline hydrochloride is a monoamine oxidase inhibitor. Warn patients against eating cheese, and using alcohol, proprietary drugs or other medication without the knowledge of the physician. When indicated, alcohol, narcotics (meperidine should be avoided), antihistamines, barbiturates, chloral hydrate, and other hypnotics, sedatives, tranquilizers, or caffeine, may be used cautiously in reduced dosage. In emergency surgery ¼ to ⅓ the usual dose of narcotics, analgesics, and other premedications should be used avoiding parenteral administration where possible. Carefully adjust dose of anesthetics to response of patient. Withdraw pargyline two weeks before elective surgery.

Warn patients about the possibility of postural hypotension. Those with angina or coronary artery disease should not increase physical activity with an improvement in well being. Pargyline may lower blood sugar.

Avoid use of enteric-coated potassium tablets, as these may induce serious or fatal small-bowel lesions consisting of stenosis with or without ulceration. These small-bowel lesions have caused obstruction, hemorrhage and perforation frequently requiring surgery. Medication should be discontinued immediately if abdominal pain, distension, nausea, vomiting or GI bleeding occurs. These products contain no added potassium salts and if added potassium intake is desired, dietary supplementation is recommended. Coated potassium tablets should be reserved for cautious use when adequate dietary supplementation is impractical. In patients with a history of allergy or asthma the possibility of sensitivity reactions should be considered.

Precautions: Measure blood pressure while patient is standing to determine antihypertensive effect. Use with caution in hyperactive or hyperexcitable persons. Such persons may show increased restlessness and agitation. Withdraw drug during acute febrile illness. Watch patients with impaired renal function for increasing drug effects or elevation of BUN and other evidence of progressive renal failure; withdraw drug if such alterations persist and progress. Use with caution in patients with liver disease. As with all new drugs, complete blood counts, urinalyses, and liver function tests should be performed periodically. With prolonged therapy, examine patients for change in color perception, visual fields and fundi. Also reported have been: blood dyscrasias including thrombocytopenia with purpura, agranulocytosis and aplastic anemia; elevations of BUN, serum uric acid, or blood sugar. Symptomatic gout may be induced. In surgical patients thiazides may reduce response to vasopressors and increase response to tubocurarine.

Adverse Reactions: Pargyline may be associated with orthostatic hypotension. Mild constipation, slight edema, dry mouth, sweating, increased appetite, arthralgia, nausea and vomiting, headache, insomnia, difficulty in micturition, nightmares, impotence, delayed ejaculation, rash, and purpura have been encountered with pargyline. Hyperexcitability, increased neuromuscular activity (muscle twitching) and other extrapyramidal symptoms have been reported in a few patients with reduced cardiac reserve.

During intensive or prolonged therapy, guard against hypochloremic alkalosis and hypokalemia (especially the latter if patient is on digitalis). Observe all patients for signs of hyponatremia ("low salt" syndrome).

Reported thiazide reactions also include anorexia, nausea, vomiting, diarrhea, headache, dizziness, paresthesia, weakness, skin rash, photosensitivity, jaundice, and pancreatitis. Nocturia has been observed with the combination.

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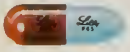
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Warning: May be habit-forming.

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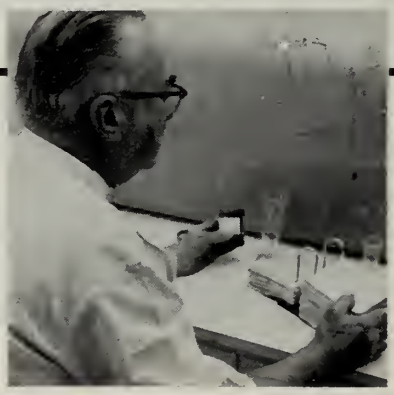
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Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported. Use of any drug in pregnancy, lactation, or in women of childbearing age requires that its potential benefits be weighed against its possible hazards.

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver-function tests advisable during protracted therapy.

Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral*—Adults: Mild and moderate anxiety and tension, 5 or 10 mg t.i.d. or q.i.d.; severe states, 20 or 25 mg t.i.d. or q.i.d. Geriatric patients: 5 mg b.i.d. to q.i.d. (See Precautions.)

Supplied: Librium® (chlordiazepoxide HCl) Capsules, 5 mg, 10 mg and 25 mg—bottles of 50. Libritabs™ (chlordiazepoxide) Tablets, 5 mg, 10 mg and 25 mg—bottles of 100. With respect to clinical activity, capsules and tablets are indistinguishable.

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LABORATORIES
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Nutley, New Jersey 07110

Virginia

MEDICAL MONTHLY

APRIL 1968

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THE FRANCIS A. COUNTWAY
LIBRARY OF MEDICINE
BOSTON
24 APR 1968



OFFICIAL PUBLICATION OF THE MEDICAL SOCIETY OF VIRGINIA

Dilantin®
(diphenylhydantoin)

PARKE-DAVIS

In untold thousands of epileptic patients... Dilantin has been, and continues to be, the bedrock of therapy.

DILANTIN is useful in the treatment of grand mal epilepsy and certain other convulsive states. Its use will prevent or greatly reduce the incidence and severity of convulsive seizures in a substantial percentage of epileptic patients, without the hypnotic and narcotizing effects of many anticonvulsant drugs.

PRECAUTIONS: Periodic examination of the blood is advisable. Nystagmus in combination with diplopia and ataxia indicates dosage should be reduced. The possibility of toxic effects during pregnancy has not been explored. ADVERSE REACTIONS: Allergic phenomena such as polyarthropathy, fever, skin eruptions, and acute generalized morbilliform eruptions with or without fever. Rarely, dermatitis goes on to exfoliation with hepatitis, and further dosage is contraindicated. Gingival hypertrophy, hirsutism, and excessive motor activity are occasionally encountered. During initial treatment, side effects may include gastric distress, nausea, weight loss, nervousness, sleeplessness, feeling of unsteadiness. Macrocytosis, megaloblastic anemia, leukopenia, granulocytopenia, thrombocytopenia, pancytopenia, agranulocytosis, and aplastic anemia have been reported. Nystagmus, lymphadenopathy, lupus erythematosus, erythema multiforme (Stevens-Johnson syndrome), and a syndrome resembling infectious mononucleosis with jaundice have occurred.

DILANTIN is supplied in several forms including Kapseals® containing 0.1 Gm. and 0.03 Gm. diphenylhydantoin sodium.

Parke, Davis & Company, Detroit, Michigan 48232

The color combinations of the banded capsules are Parke-Davis trademarks. The orange-banded white capsule identifies Parke-Davis 0.1 Gm. diphenylhydantoin sodium; the pink-banded white capsule 0.03 Gm. diphenylhydantoin sodium.

PARKE-DAVIS 015R67



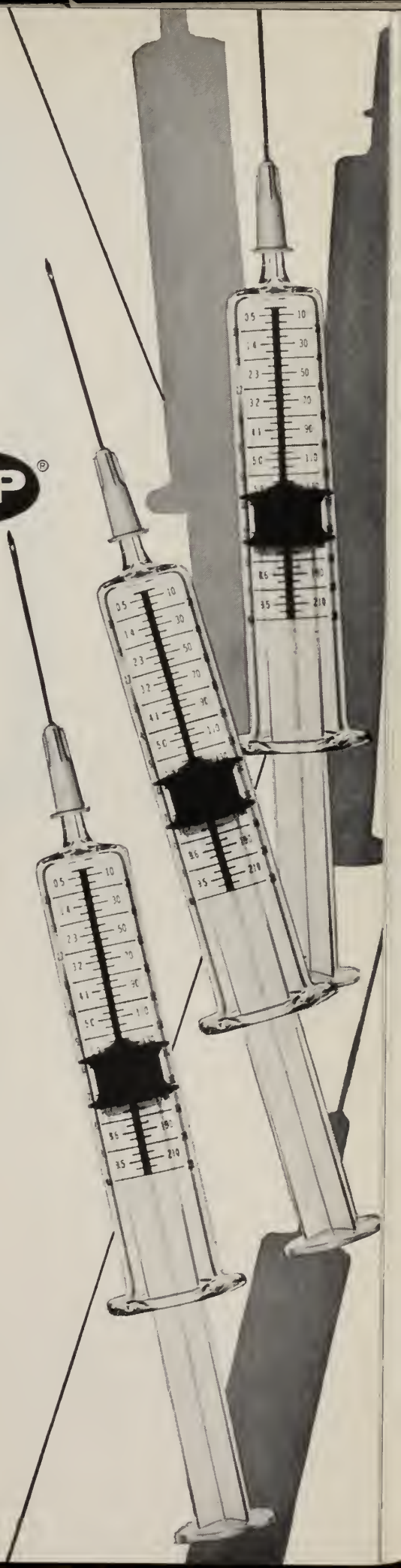
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(50 mg. per ml.)

BSP®



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b.i.d.

The sensible schedule that covers the patient day and night

If your objective in the use of a broad-spectrum antibiotic is prolonged action, with high blood levels, then you know why b.i.d. DECLOMYCIN is considered to be a sensible dosage schedule.

The maintenance dosage of DECLOMYCIN can be kept at this convenient schedule because of its unusually high effective blood and tissue levels.

The b.i.d. dosage of DECLOMYCIN gives you the comfortable assurance that the patient is well-covered, *day and night.*

In clinical practice, blood levels produced by a therapeutic dose of DECLOMYCIN are high, prolonged, and effective; because of high serum binding and slow renal clearance. And if there's a broader susceptibility pattern of organisms, we've yet to see it.

There is no need to give higher daily dosage than 300 mg b.i.d., except in venereal diseases and Eaton Agent pneumonia.

DECLOMYCIN®
DEMETHYLCHLORTETRACYCLINE

Lederle

Prescribing information on next page.

b.i.d. The sensible schedule that covers the patient day and night

DECLOMYCIN Demethylchlortetracycline should be equally or more effective therapeutically than other tetracyclines when the offending organisms are tetracycline-sensitive.

Contraindication: History of hypersensitivity to demethylchlortetracycline.

Warning—In renal impairment, usual doses may lead to excessive accumulation and liver toxicity. Under such conditions, lower than usual doses are indicated, and, if therapy is prolonged, serum level determinations may be advisable. A photodynamic reaction to natural or artificial sunlight has been observed. Small amounts of drug and short exposure may produce an exaggerated sunburn reaction which may range from erythema to severe skin manifestations. In a smaller proportion, photoallergic reactions have been reported. Patients should avoid direct exposure to sunlight and discontinue drug at the first evidence of skin discomfort. Necessary subsequent courses of treatment with tetracyclines should be carefully observed.

Precautions—Overgrowth of nonsusceptible organisms may occure. Constant observation is essential. If new infections appear, appropriate measures should be taken. In infants, increased intracranial pressure with bulging fontanels has been observed. All signs and symptoms have disappeared rapidly upon cessation of treatment.

Side Effects—Gastrointestinal system—anorexia, nausea, vomiting, diarrhea, stomatitis, glossitis, enterocolitis, pruritus ani. Skin—maculopapular and erythematous rashes. A rare case of exfoliative dermatitis has been reported. Photosensitivity; onycholysis and discoloration of the nails (rare). Kidney—rise in BUN. apparently dose related. Transient increase in urinary output, sometimes accompanied by thirst (rare). Hypersensitivity reactions—urticaria, angioneurotic edema, anaphylaxis. Teeth— dental staining (yellow-brown) in children of mothers given this drug during the latter half of pregnancy, and in children given the drug during the neonatal period, infancy and early childhood. Enamel hypoplasia has been seen in a few children. If adverse reaction or idiosyncrasy occurs discontinue medication and institute appropriate therapy.

Average Adult Daily Dosage: 150 mg q.i.d. or 300 mg b.i.d. Should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products. Treatment of streptococcal infections should continue for 10 days, even though symptoms have subsided.

In the treatment of syphilis a dosage schedule of a total of 12 to 18 Gm. given in equally divided doses over a period of 10 to 15 days should be followed. Close follow-up observation of the patient is recommended, including appropriate laboratory tests, since demethylchlortetracycline has not had adequate evaluation in all stages of syphilis. Spinal fluid examination should be included as part of this follow-up.

Acute gonococcal anterior urethritis in males has been treated effectively with a single dose of 600-900 mg. of DECLOMYCIN Demethylchlortetracycline. Individuals unable to tolerate large single doses due to gastrointestinal side effects may be treated with 150 mg. every 6 hours for a minimum of 4 doses or 300 mg. every 12 hours for a minimum of 2 doses. Females should be treated with a dosage of 150 mg. every 6 hours or 300 mg. every 12 hours until a cure is effected.

Primary Atypical Pneumonia (Eaton Agent): The average adult daily dosage is 900 mg. in 3 divided doses for six days.

LEDERLE LABORATORIES, A Division of American Cyanamid Company, Pearl River, N.Y.

Lederle

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(SYRUP OF CHLORAL HYDRATE)

A palatable chloral hydrate syrup

containing 10 grains in each teaspoonful.

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Where do you get the money . . . .

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- . . **that you normally spend on shelter?**
- . . **that it takes to maintain your office?**

There is no question how your income will continue if you enroll in the DISABILITY INCOME PLAN which is sponsored and recommended by THE MEDICAL SOCIETY OF VIRGINIA.

With these outstanding features:

- ✓ Benefits paid directly to you. You spend the money any way you want;
- ✓ This plan is yours until age 75; (Plan A or Plan B)
- ✓ Under Plan A—Benefits paid to age 65 for sickness—lifetime for accident.

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Protect your earnings and ability to pay. Get complete details during the Open Enrollment Period—now in progress.

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Dave,

I have enough problems without having to worry about how I'll pay the rent, the grocery bills, or keep the office open if I'm disabled. Please rush to me, without obligation, all the facts on the Disability Income Program for physicians and surgeons, underwritten by Insurance Company of North America, Phila., Pa.

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Street

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(City) (State) (Zip)

Underwritten by The Insurance Company of North America, Philadelphia, Pa.

Rx: MONEY

For relief from the worry and expense brought on by accident and sickness disability.

When doctors are disabled and prevented from practicing, expenses mount up fast. They face not only the bills associated with today's costly medical treatment, but the great expense of maintaining an office and full staff as well. That's why the cost of just one month's disability often runs into thousands of dollars!

Your Medical Society of Virginia knows this. That's why they have sponsored two plans that offer the *standard* remedy:

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- A Professional OVERHEAD EXPENSE Plan
which pays fixed office expenses when you're disabled and prevented from practicing due to accident or sickness.

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which pays the high costs of medical treatment associated with accident and sickness disability.

Both of these Plans are sensibly priced because of your Association's sponsorship.
And either or both of them can go to work for you, today . . . if you call us now. Find out for yourself why your Society has selected these insurance plans as the best available to its Members. For more information, write or call collect. There is no obligation, of course.

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Medical Arts Building
Roanoke, Virginia 24011 Phone: 344-5000

Both Plans underwritten by

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TROCINATE® 400 MG.

BRAND THIPHENAMIL HCl.

The relief received from the first Trocinate 400 mg. tablet is so prompt that the discomfort of diarrhea ceases to be a bother. May be repeated every four hours.

Upon request, a supply of Trocinate 400 mg. with literature will be sent to physicians for their personal use.

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Manufacturers of ethical pharmaceuticals since 1856

Whenever the diet is faulty, the appetite poor, or the loss of food is excessive

through vomiting or diarrhea—

Valentine's MEAT EXTRACT

stimulates the appetite,

increases the flow of digestive juices,

provides: supplementary amounts of vitamins, minerals and soluble proteins,

extra-dietary vitamin B_{12},

protective quantities of potassium, in a palatable and readily assimilated form.

Supplied in bottles of 2 or 6 fluidounces.

DOSAGE *is 1 teaspoonful two or three times daily; two or three times this amount for potassium therapy. Dilute with two or more equal volumes of water.*

VALENTINE Company, Inc.
RICHMOND 21, VIRGINIA

Abbott Antihypertensive Building Blocks

A simplified approach to the practical management of hypertension



Enduron: (methyclothiazide) A basic building block for mild hypertensives



Excellent day-long Na^+ output, yet easy on the K^+

Enduron provides an excellent starting therapy. Your patient's sodium excretion is greatly enhanced. Yet potassium loss is low.

The therapeutic action is smooth, and persists for a full 24 hours. With Enduron you can prescribe convenient once-a-day dosage without skimping your patients on day-long thiazide effectiveness.

Of course, as with all thiazides, supplemental dietary potassium should also be considered.

Use Enduron as a basic therapy in patients with mild to moderate hypertension. A single 5-mg. tablet each day is ample in most cases.

Once a day, every day

ENDURON

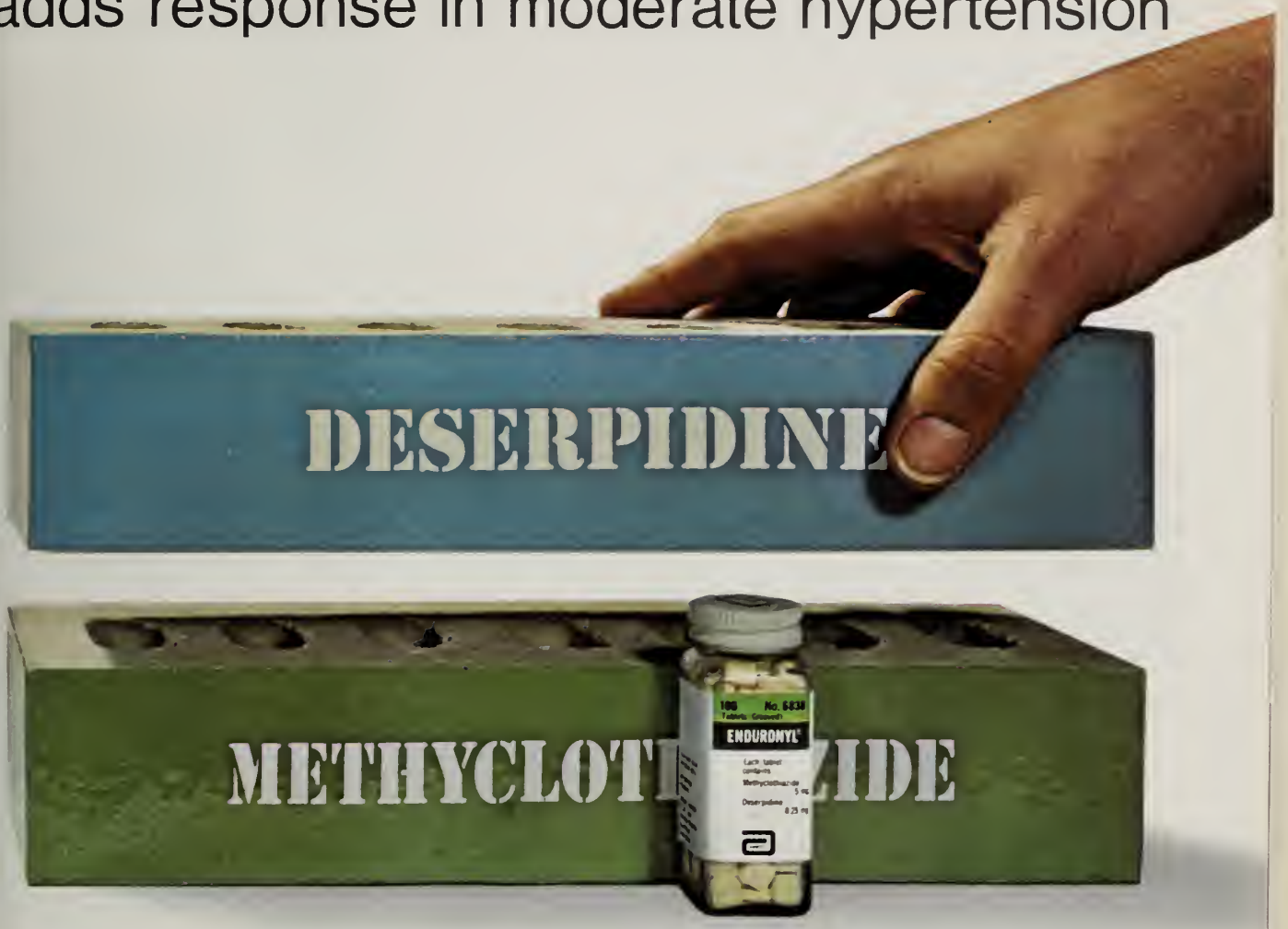
METHYCLOTHIAZIDE

MILD TO MODERATE TO SEVERE



See Brief Summary on final page of advertisement

Enduronyl: Its deserpidine component adds response in moderate hypertension



Less frequent rauwolfia side effects than with reserpine

When you wish to build further response, consider shifting to Enduronyl.

Enduronyl adds a building block of *deserpidine*. This is a purified rauwolfia alkaloid available only from Abbott. It adds good antihypertensive and tranquilizing activity. Yet its incidence of untoward effects, particularly lethargy and depression, is lower than with reserpine.

Enduronyl is available plain or Forte. The latter provides its variation where most helpful, by doubling the deserpidine.

Use Enduronyl for patients throughout the broad range of mild to moderately severe hypertension.

Once a day, every day

ENDURONYL®

METHYCLOTHIAZIDE 5 mg. with
DESERPIDINE 0.25 mg. or (FORTE) 0.5 mg.

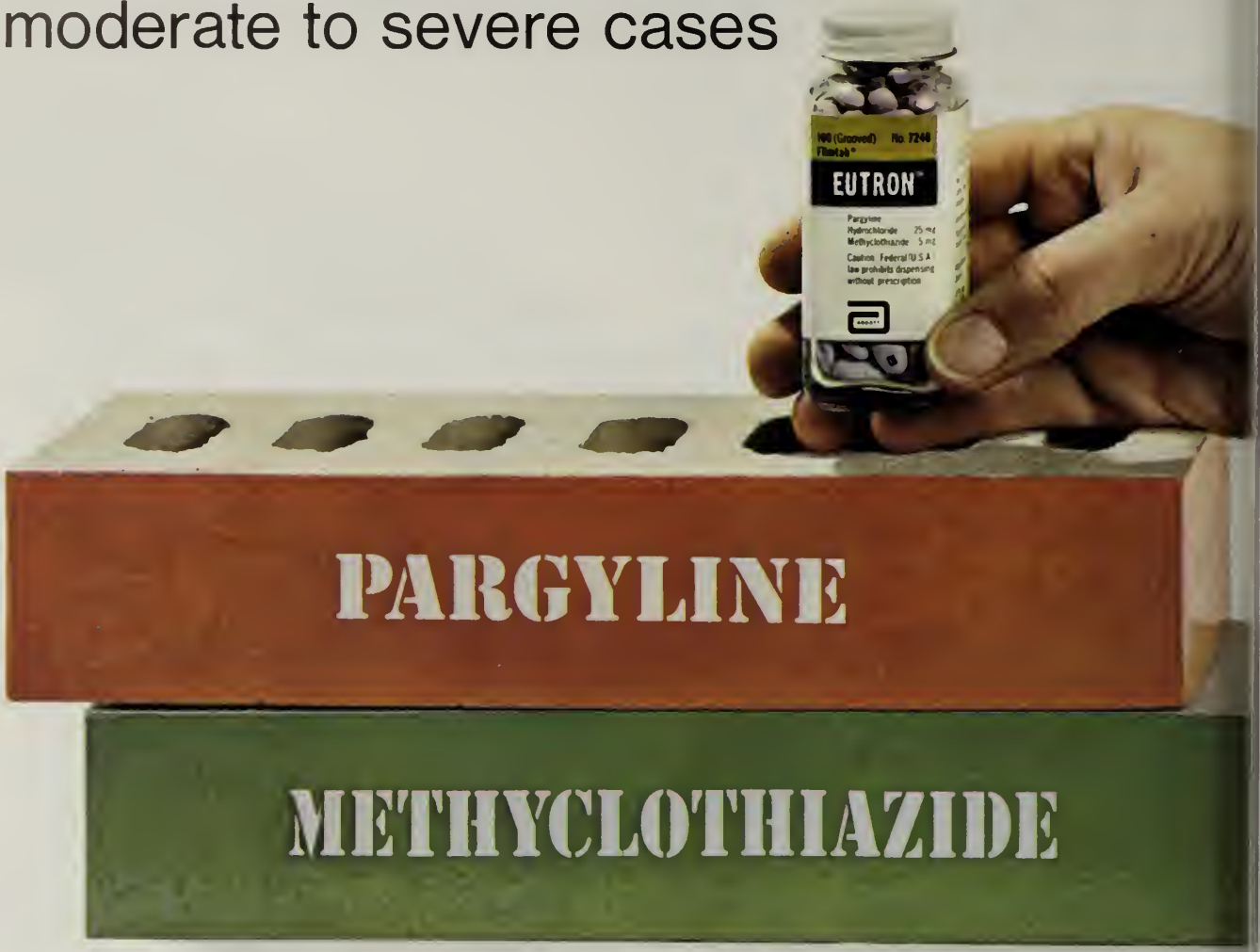
MILD TO MODERATE TO SEVERE



See Brief Summary on final page of advertisement

801094

Eutron: A unique combination for handling moderate to severe cases



Affords almost uniform diastolic reduction in all body positions

Eutron lowers diastolic pressures nearly equally, whether your patient is standing up or lying down.

Thus, in clinical trials, average *standing* diastolic readings were reduced from 112 pre-treatment to 90 post-treatment; *sitting* from 115 to 95; and *recumbent* from 112 to 94.

Note that following Eutron, the diastolic reductions were nearly alike in all three body positions.

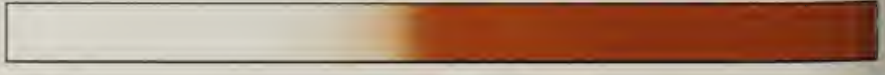
Use Eutron for managing your moderate to severe cases. Its building blocks enhance each other; hence lesser doses often suffice.

Once a day, every day

EUTRON™

PARGYLINE HYDROCHLORIDE 25 mg.
with METHYCLOTHIAZIDE 5 mg.

MILD TO MODERATE TO SEVERE



See Brief Summary on final page of advertisement

801094

ENDURON® | ENDURONYL®

METHYCLOTHIAZIDE

Each tablet contains Methyclothiazide 5 mg. with Deserpidine 0.25 mg. or 0.5 mg.

Indications: Edema and mild to moderate hypertension (Enduron), and mild to moderately severe hypertension (Enduronyl). More potent agents, if added, can be given at reduced dosage.

Contraindications: Sensitivity to thiazides; severe renal disease (except nephrosis) or shutdown; severe hepatic disease or impending hepatic coma (hepatic coma due to hypokalemia has been reported in patients on thiazides). Do not use Enduronyl in severe mental depression, suicidal tendencies, active peptic ulcer, or ulcerative colitis.

Warnings: Consider possible sensitivity where there is history of allergy or asthma. If added potassium is indicated, dietary supplementation is recommended. Reserve enteric-coated potassium tablets for cautious use only when necessary, as they may induce serious or fatal small bowel lesions (stenosis with or without ulceration), cause obstruction, hemorrhage, and perforation often requiring surgery; discontinue them immediately if abdominal pain, distention, nausea, vomiting, or g.i. bleeding occurs. Neither Enduron nor Enduronyl contains added potassium.

Precautions: Use thiazides cautiously in severe renal dysfunction, impaired hepatic function or progressive liver disease; also in pregnancy (bone marrow depression, thrombocytopenia, and altered carbohydrate metabolism have been reported in certain newborn). In surgery, thiazides may reduce response to vasopressors, and increase response to tubocurarine. Antihypertensive response may be enhanced following sympathectomy. Watch for electrolyte imbalance (e.g., hyponatremia) in all patients. In hypokalemia (especially in digitalized patients) give supplemental potassium. In hypochloremic alkalosis, give supplemental chloride.

Use rauwolfias with caution in patients with history of peptic ulcer. Rauwolfias with anesthetics may produce hypotension and bradycardia. Discontinue Enduronyl two weeks before elective surgery. Consider vagal blocking agents during emergency surgery. In epilepsy, adjust anticonvulsant dosage. In electroshock, shorten stimulus strength and duration. In occasional patients with depressive tendencies, rauwolfias may precipitate severe mental depression that usually disappears when drug is stopped.

Adverse Reactions: Thiazide reaction include blood dyscrasias (thrombocytopenia with purpura, agranulocytosis, aplastic anemia); elevation of BUN, serum uric acid or blood sugar; anorexia, nausea, vomiting, diarrhea, headache, dizziness, paresthesia, weakness, skin rash, photosensitivity, jaundice, symtomatic gout, and pancreatitis. Cutaneous vasculitis in the elderly has been reported with other thiazides. Adverse effects with deserpidine are qualitatively similar to those with reserpine, but their incidence is lower. These include nasal stuffiness, abdominal cramps or diarrhea, nausea, headache, weight gain, reduced libido and potency, peptic ulcer aggravation, epistaxis, skin eruption, asthma in susceptible patients, electrolyte imbalance, excessive salivation, and a reversible Parkinson's syndrome. Excessive drowsiness, fatigue, weakness, and nightmares may signal mental depression. Thrombocytopenia, purpura, and a symptom manifested by dull sensorium, deafness, uveitis, glaucoma, and optic atrophy are rare allergic reactions to other rauwolfias. Hypotension from antihypertensive agents may precipitate angina attacks in susceptible individuals. Usually adverse reactions disappear when drug is withdrawn.

EUTRON™

Each tablet contains Pargyline Hydrochloride 25 mg. with Methyclothiazide 5 mg.

Indications—Moderate to severe hypertension.

Contraindications—Pheochromocytoma, paranoid schizophrenia, hyperthyroidism and advanced renal failure. Not recommended in malignant hypertension, children under 12, pregnant patients.

Do not use with: centrally or peripherally acting sympathomimetic drugs; foods high in tyramine (e.g., aged and natural cheeses); parenteral reserpine or guanethidine; imipramine, amitriptyline, desipramine, nortriptyline or their analogues; other monoamine oxidase inhibitors; methyldopa or dopamine; separate Eutron and these agents by two weeks.

Sensitivity to thiazides; severe renal disease (except nephrosis) or shutdown; severe hepatic disease; impending hepatic coma from thiazide-induced hypokalemia.

Warnings—Patients: 1. No other drugs (particularly "cold preparations" and antihistamines), cheese or alcohol without physician's consent. 2. Promptly report orthostatic symptoms, severe headache, other unusual symptoms. 3. Angina pectoris or coronary artery disease patients must not increase physical activity with improved anginal symptoms or well-being.

Physicians: 1. Use antihistamines, hypnotics, sedatives, tranquilizers and narcotics (meperidine contraindicated) cautiously in reduced doses. 2. Stop Eutron two or more weeks before elective surgery; in emergency surgery reduce premedication (narcotics, sedatives, analgesics, etc.) to 1/4 to 1/5; carefully adjust anesthetic dosage to patient response. 3. Use cautiously in advanced renal failure. 4. Pargyline may induce hypoglycemia. 5. Consider possible sensitivity reactions when a history of allergy or asthma is present. 6. If potassium is indicated, dietary supplement is recommended; enteric-coated potassium tablets may induce serious or fatal small bowel lesions (stenosis with or without ulceration), cause obstruction, hemorrhage, and perforation frequently requiring surgery; discontinue medication immediately if abdominal pain, distention, nausea, vomiting or gastrointestinal bleeding occurs; Eutron does not contain added potassium. 7. Possible systemic lupus erythematosus has been reported for thiazides.

Precautions—Pargyline: Use cautiously at reduced dosage: caffeine, alcohol, antihistamines, barbiturates, chloral hydrate, other hypnotics, sedatives, tranquilizers, narcotics. Periodically do urinalyses, blood counts, liver function tests, etc. Use with caution in liver disease. Watch for orthostatic hypotension, especially in impaired circulation (e.g., angina pectoris, coronary artery disease, cerebral arteriosclerosis); also, augmented hypotension in concomitant febrile illnesses. Reduce or discontinue if hypotension is severe. In impaired renal function watch for cumulative drug effects, elevated BUN and other evidence of progressive renal failure; withdraw drug if these persist. In surgery increased central depressant response (hypotension and increased sedative effect) can be controlled by (1) discontinuing at least two weeks prior; (2) in emergency surgery lowering dose of premedication; (3) when necessary, administering a vasopressor. Do not use in hyperactive and hyperexcitable patients. Pargyline may unmask severe psychotic symptoms where emotional problems pre-exist. Use cautiously in Parkinsonism, especially with antiparkinsonian agents. In prolonged therapy, examine for change in color perception, visual fields, fundi and visual acuity. Also, prolonged therapy has made certain patients refractory to nerve blocking effects of local anesthetics.

Methyclothiazide: Use cautiously in severe renal dysfunction, impaired hepatic function or progressive liver disease; also in pregnancy (bone marrow depression, thrombocytopenia, and altered carbohydrate metabolism have been reported in certain newborn). In surgery thiazide may reduce vasopressor response and increase tubocurarine response. Antihypertensive response may be enhanced following sympathectomy. Watch for electrolyte imbalance (e.g., hyponatremia). Give supplemental chloride if hypochloremic alkalosis occurs and supplemental potassium if hypokalemia occurs (especially in digitalized patients). Thiazides may decrease serum P.B.I. without signs of thyroid disturbance.

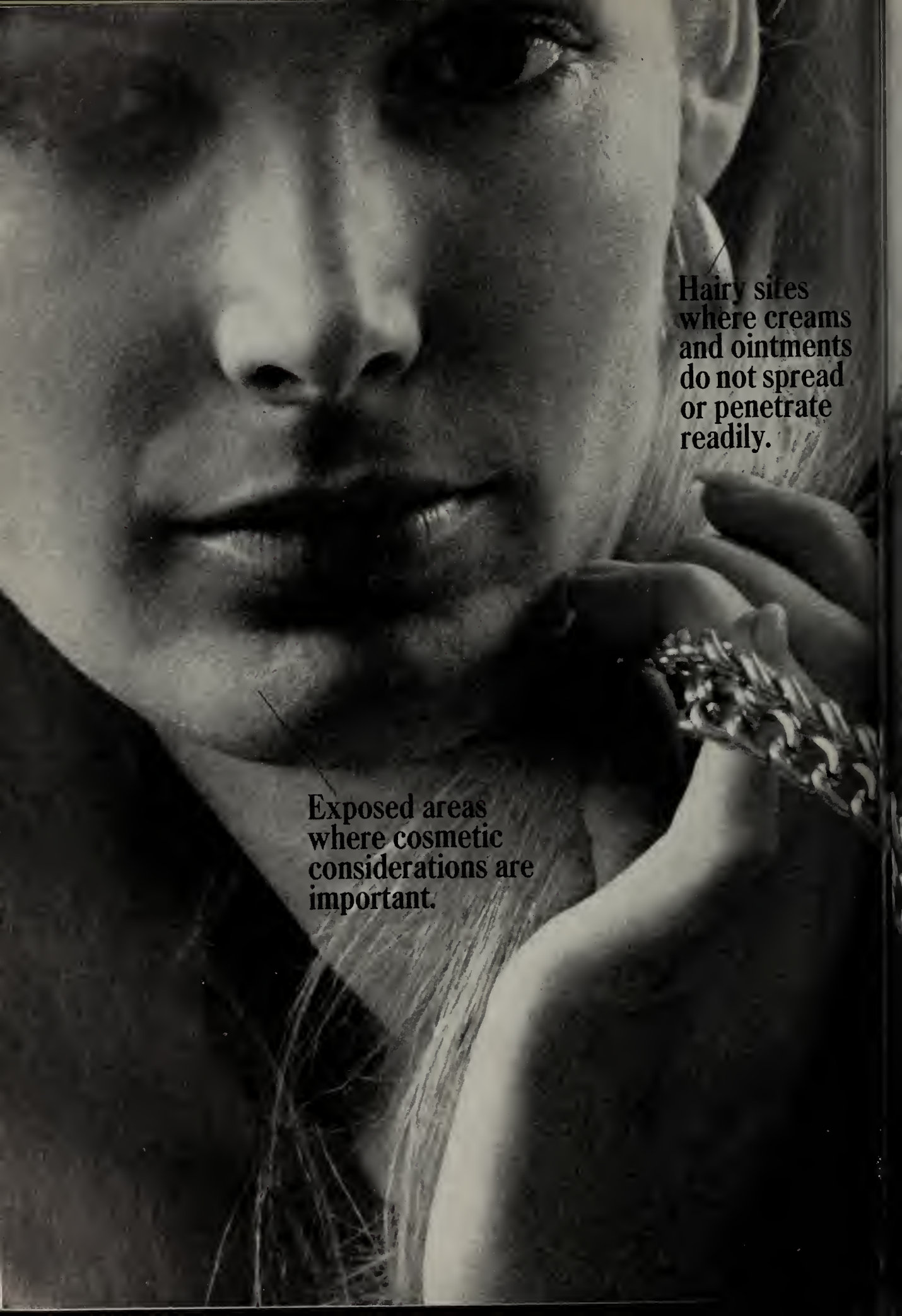
Adverse Reactions — Pargyline: Orthostatic hypotension and associated symptoms, mild constipation, fluid retention, edema, dry mouth, sweating, increased appetite, arthralgia, nausea, vomiting, headache, insomnia, difficult in micturition, nightmares, impotence, delayed ejaculation, rash, purpura, weight gain, hyperexcitability, increased neuromuscular activity and other extrapyramidal symptoms. Drug fever is extremely rare. Reduction in blood sugar and hypoglycemic effects are possible. Congestive heart failure has been reported in a few patients with reduced cardiac reserve.

Methyclothiazide: Blood dyscrasias (thrombocytopenia with purpura, agranulocytosis, aplastic anemia); elevation of BUN, blood sugar or serum uric acid (gout may be induced); anorexia, nausea, vomiting, diarrhea, headache, dizziness, paresthesia, weakness, skin rash, photosensitivity, jaundice and pancreatitis. Cutaneous vasculitis in elderly patients has been reported with other thiazides.

If side effects are severe or persist, reduce dosage or withdraw drug. 804438R

ABBOTT

TM-TRADEMARK


Hairy sites where creams and ointments do not spread or penetrate readily.
Exposed areas where cosmetic considerations are important.

An invisible topical

for many dermatologic problems



See last page for contraindications, precautions and side effects.

Structurally unique topical steroid in propylene glycol vehicle produces rapid response in many dermatoses.

Synalar Solution produces rapid antiinflammatory, antipruritic action through its unique topical corticosteroid, fluocinolone acetonide. The propylene glycol vehicle provides additional benefits for therapy at problem sites where it is difficult to achieve contact between the lesion and medication—or where creams or ointments may make the lesions worse. Synalar Solution has proved particularly valuable in the symptomatic treatment of seborrheic dermatitis of the scalp, nasolabial folds, eyebrows, ears, and in flexural folds such as the axillae, inframammary, umbilical and anocrural areas. It has also been reported to achieve excellent results in the adjunctive management of atopic dermatitis, contact dermatitis, neurodermatitis, nummular eczema, psoriasis, and sweat retention syndromes in these problem sites.

Penetrates the hairy sites.

In many areas of the body, hair gets in the way of treating the underlying dermatitis. The propylene glycol vehicle of Synalar Solution permits penetration and dispersion at sites where creams and ointments do not readily penetrate. May be applied without matting of hair.

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Propylene glycol is strongly hygroscopic and is especially useful where sweat retention is a problem. Its low surface tension permits easy spreadability in difficult-to-treat body areas. A number of studies have also shown that propylene glycol has inherent antimicrobial activity.



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The propylene glycol vehicle of Synalar Solution possesses many useful cosmetic properties. Clear and greaseless, it is not sticky or messy, will not stain clothing or skin. In exposed areas of the body where cosmetic appeal is important, Synalar Solution shows nothing but results.

Economical—a little goes a long way.

Because of the properties of propylene glycol and the milligram potency of fluocinolone acetonide, a small quantity of Synalar Solution goes a long way. Also, the prescription price of a 20 cc. plastic squeeze bottle of Synalar Solution is surprisingly low. Thus, your patients obtain economy with the proved efficacy of a potent, truly advanced topical corticosteroid.

Contraindications: Tuberculous, fungal, and most viral lesions of the skin (including herpes simplex, vaccinia, and varicella). Not for ophthalmic use. Contraindicated in individuals with a history of hypersensitivity to any of the components.

Precautions: In some patients with dry lesions, the solution may increase dryness, scaling, or itching. Application to denuded or fissured areas, such as genital or perianal sites, may produce a burning or stinging sensation. If this persists and dermatitis does not improve, discontinue medication. Although propylene glycol has antiseptic activity, there should be careful initial evaluation and follow-up of infected sites. Incomplete response or exacerbation of lesions may be due to true infection, which requires susceptibility testing and appropriate therapy. On the other hand, saprophytic or low grade infections may clear spontaneously under the influence of Synalar Solution alone. Where severe local infection or systemic infection exists, the use of systemic antibiotics should be considered, based on susceptibility testing. While topical steroids have not been reported to have adverse effect on pregnancy, the safety of their use on pregnant females has not absolutely been established. Therefore, they should not be used extensively on pregnant patients, in large amounts, or for prolonged periods of time.

Side Effects: Side effects are not encountered ordinarily with topically applied corticosteroids. As with all drugs, however, a few patients may react unfavorably to Synalar under certain conditions.

Availability: Synalar (fluocinolone acetonide) Solution 0.01% in a propylene glycol vehicle with citric acid as preservative. 20 and 60 cc. plastic squeeze bottles. *Also available:* Synalar (fluocinolone acetonide) Cream 0.025%—5, 15 and 60 Gm. tubes and 425 Gm. jars. Cream 0.01%—15, 45 and 60 Gm. tubes and 120 Gm. jars. Ointment 0.025%—15 and 60 Gm. tubes. Neo-Synalar® (neomycin sulfate 0.5% [0.35% neomycin base], fluocinolone acetonide 0.025%) Cream—5, 15 and 60 Gm. tubes.

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CONTRAINDICATIONS: Glaucoma, urethral or ureteral spasm, recent myocardial infarction, severe coronary heart disease, epilepsy. Should not be given within two weeks of treatment with a monoamine oxidase inhibitor.

RELATIVE CONTRAINDICATIONS: (1) Patients with a history of paroxysmal tachycardia. (2) Patients receiving concomitant therapy with thyroid, anticholinergics or sympathomimetics may experience potentiation of effects of these drugs. (3) Safety in pregnancy has not been established. (4) Perform liver function studies in patients suspect of having hepatic disease.

PRECAUTIONS: (1) Desipramine hydrochloride should not be substituted for hospitalization when risk of suicide or homicide is considered grave. Suicidal ingestion of large doses may be fatal. (2) If serious adverse effects occur, reduce dosage or alter treatment. (3) In patients with manic-depressive illness a hypomanic state may be induced. (4) Discontinue drug as soon as possible prior to elective surgery.

ADVERSE EFFECTS: The following side effects hav been encountered: dry mouth, constipation, dizz ness, palpitation, delayed urination, agitation ar stimulation ("jumpiness," "nervousness," "anxiety "insomnia"), bad taste, sensory illusion, tinnitu sweating, drowsiness, headache, hypotensio (orthostatic), flushing, nausea, cramps, weaknes blurred vision and mydriasis, rash, tremor, allerg (general), altered liver function, ataxia and extr pyramidal signs, agranulocytosis.

Additional side effects more recently reporte include: seizures, eosinophilia, confusional stat with hallucinations, purpura, photosensitivity, gala torrhea, gynecomastia, and impotence. Side effec which could occur (analogy to related drugs) i clude weight gain, heartburn, anorexia, and har and arm paresthesias.

DOSAGE: Optimal results are obtained at a dosa of 50 mg. t.i.d. (150 mg./day).

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SPHENOID SINUS—most difficult to diagnose. Headache or sense of pressure over vertex. May be pain in ear or mastoid region.



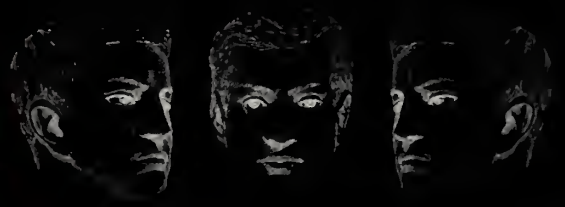
MAXILLARY SINUS—headache and tenderness under eyes at top of cheekbones, usually starting in early afternoon. Possible swelling of cheek. Pain in jaw and back teeth.
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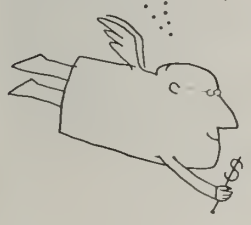
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DOSAGE

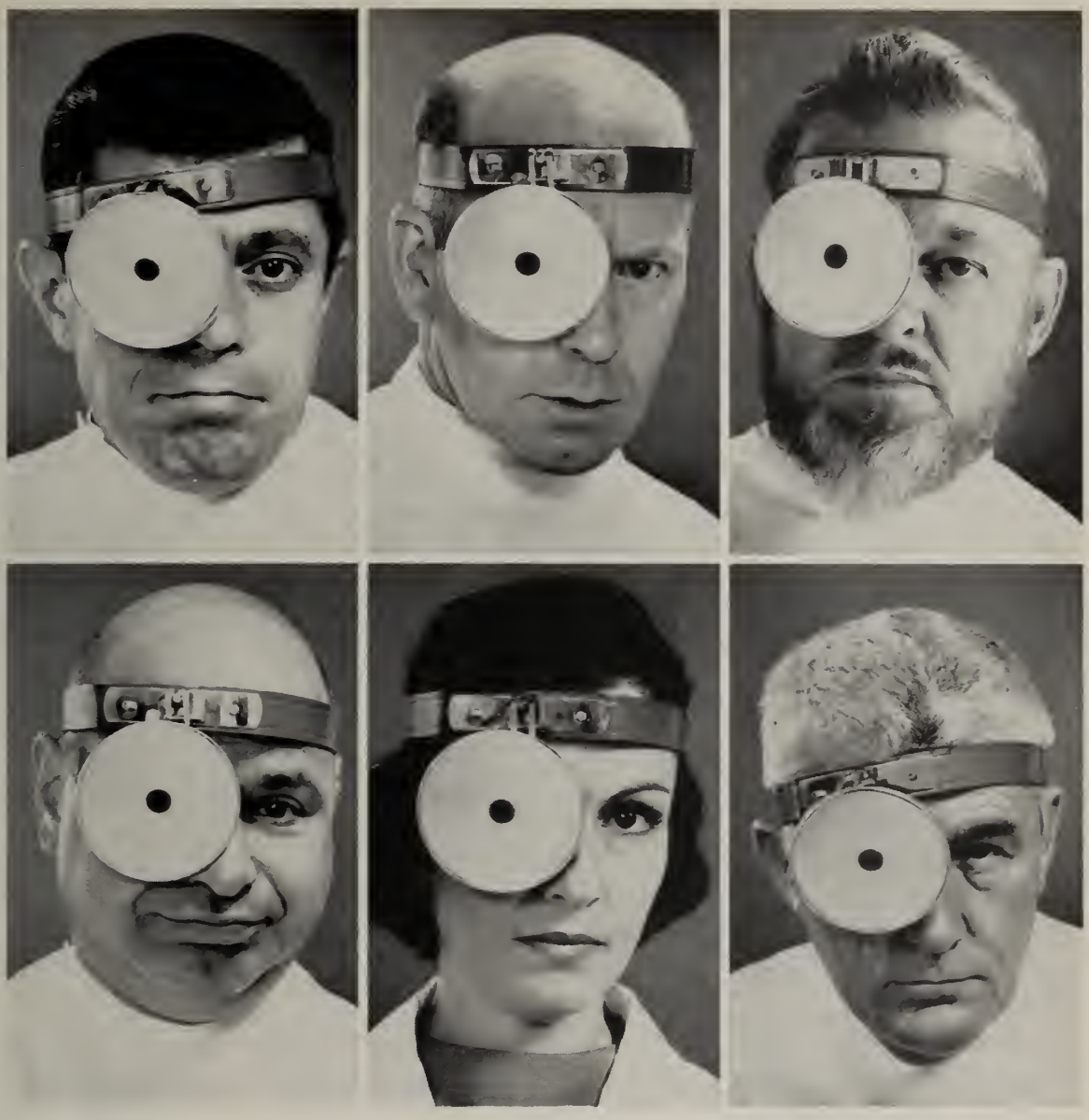
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when you want to provide the combined benefits of two accepted agents in the treatment of mild to moderate hypertension. (Contraindications: history of mental depression, hypersensitivity, and most cases of severe renal or hepatic diseases.)

when you want to prescribe therapy that is generally well tolerated.
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Indications: Hypertension. **Contraindications:** History of mental depression, hypersensitivity, and most cases of severe renal or hepatic diseases. **Warning:** With the administration of enteric-coated potassium supplements, which should be used only when adequate dietary supplementation is not practical, the possibility of small-bowel lesions (obstruction, hemorrhage, and perforation) should be kept in mind. Surgery for these lesions has frequently been required and deaths have occurred. Discontinue coated potassium-containing formulations immediately if abdominal pain, distention, nausea, vomiting, or gastrointestinal bleeding occur. Discontinue 1 week before electroshock therapy, and if depression or peptic ulcer occurs. **Use in pregnancy:** Regroton should be used in pregnant patients or in women of childbearing potential only when, in the judgment of a physician, its use is deemed essential to the welfare of the patients; adverse reactions (thrombocytopenia, hyperbilirubinemia, altered carbohydrate metabolism, etc.) are potential problems in the newborn. **Precautions:** Antihypertensive therapy with Regroton should always be initiated cautiously in post-sympathectomy patients and in patients receiving ganglionic blocking agents, other potent antihypertensive drugs, or curare. Reduce dosage of concomitant antihypertensive agents by at least one-half. To avoid hypotension during surgery, discontinue Regroton therapy two weeks prior to elective surgical procedures. In emergency surgery, use, if needed, anticholinergic or adrenergic drugs or other supportive measures as indicated. Because of the possibility of progression of renal damage, periodic kidney function tests are indicated. Discontinue if the BUN rises or liver dysfunction is aggravated. Hepatic coma may be precipitated. Electrolyte imbalance, sodium and/or potassium depletion may occur. If potassium depletion should occur during therapy, Regroton should be discontinued and potassium supplements given, provided the patient does not have marked oliguria. Take particular care in cirrhosis or severe ischemic heart disease and in patients receiving corticosteroids, ACTH, or digitalis. Salt restriction is not recommended. Use cautiously in patients with ulcerative colitis or gallstones (biliary colic may be precipitated). Bronchial asthma may occur in susceptible patients. **Adverse Reactions:** The drug is generally well tolerated. The most frequent side effects are nausea, gastric irritation, vomiting, diarrhea, constipation, muscle cramps, headache, dizziness and acute gout. Other potential side effects include angina pectoris, anxiety, depression, bradycardia and ectopic cardiac rhythms (especially when used with digitalis), drowsiness, dull sensorium, hyperglycemia and glycosuria, hyperuricemia, lassitude, restlessness, transient myopia, impotence or dysuria, orthostatic hypotension which may be potentiated when chlorthalidone is combined with alcohol, barbiturates or narcotics, leukopenia, aplastic anemia, skin rashes, thrombocytopenia, agranulocytosis, nasal stuffiness, increased gastric secretions, nightmare, purpura, urticaria, ecchymosis, weakness, uveitis, optic atrophy and glaucoma, and pruritus. Eruptions and/or flushing of the skin, a reversible paralysis agitans-like syndrome, blurred vision, conjunctival injection, increased susceptibility to colds, dyspnea, weight gain, decreased libido, dryness of the mouth, deafness, anorexia, and pancreatitis when epigastric pain or unexplained G.I. symptoms develop after prolonged administration. Jaundice, xanthopsia, paresthesia, photosensitization and necrotizing angiitis are possible. **Average Dosage:** One tablet daily with breakfast. **Availability:** Pink, single-scored tablets in bottles of 100 and 1000. **For details, see complete Prescribing Information.** (B)46-600-B

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Equagesic®

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Contraindications: History of sensitivity or severe intolerance to aspirin or meprobamate.

Warnings: USE IN PREGNANCY: Safety for use during pregnancy or lactation has not been established; therefore, it should be used in pregnant patients or women of childbearing age only when the physician judges its use essential to the patient's welfare.

Precautions: Keep out of reach of children. Not recommended for patients 12 years old or less. Carefully supervise dose and amounts prescribed, especially for patients prone to overdose themselves. Excessive prolonged use of meprobamate may result in dependence or habituation in susceptible persons—as alcoholics, ex-addicts, severe psychoneurotics. Withdraw gradually after prolonged high dosage to avoid possibly severe withdrawal reactions including epileptiform seizures. Warn patients of possible reduced alcohol tolerance. If drowsiness, ataxia or visual disturbances (impairment of accommodation and visual acuity) occur, reduce dose. If symptoms persist, caution patients against operating machinery or driving. After meprobamate overdose, prompt sleep, reduction of blood pressure, pulse and respiratory rates to basal levels, and hyperventilation are reported. Give cautiously to patients with suicidal tendencies. Treat attempted suicide (has resulted in coma, shock, vasomotor and respiratory collapse and anuria) with immediate gastric lavage and appropriate supportive therapy (CNS stimulants and pressor amines as indicated).

Side Effects: Ethoheptazine and aspirin may occasionally cause nausea, vomiting, epigastric distress, and rarely dizziness. Overdosage may result in CNS depression (drowsiness and lightheadedness) or CNS stimulation and salicylate intoxication (requires induced vomiting or gastric lavage, specific parenteral electrolyte therapy for ketoacidosis and dehydration, and observation for hypoprothrombinemic hemorrhage [usually requires whole blood transfusions]). Meprobamate may cause drowsiness, ataxia and rarely allergic or idiosyncratic reactions. These reactions, sometimes severe, can develop in patients receiving only 1 to 4 doses who have had no previous contact with meprobamate. Mild reactions are characterized by urticarial or erythematous maculopapular rash. Acute nonthrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever have been reported. If allergic reaction occurs, meprobamate should be stopped and not reinstituted. Severe reactions, observed very rarely, include angioneurotic edema, bronchial spasms, fever, fainting spells, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case) and hyperthermia. Treat symptomatically such as with epinephrine, antihistamine and possibly hydrocortisone. A few cases of leucopenia, usually transient, have been reported following continuous use. Rarely, cases of aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis, and hemolytic anemia have been reported; almost always, in the presence of known toxic agents.

Composition: 150 mg. meprobamate, 75 mg. ethoheptazine citrate and 250 mg. aspirin per tablet.

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The Virginia
MEDICAL MONTHLY

April, 1968
Vol. 95, No. 4
Whole No. 1391

Guest Editorial . . .

The Extended Care Facility and Medicare

MEDICARE has been thrust on our unsuspecting professional lives like a perpetual epidemic, causing symptoms of malaise and depres-pression. It is snarled with ubiquitous regulations, red tape and governmental snooping into our professional behavior. It has increased the credibility gap between what our administration has offered to the public and what is our capacity as physicians to deliver. In short, it has promised more than any society can be expected to offer to its sick, or well, for that matter. However, whether we like it or not, the Great Society operates in this fashion not only in health and welfare, but in foreign policy and domestic policy alike. It is not likely to change, and since it has been given the respectability of law, we have no alternative but to obey.

In this context I wish to present a few pertinent remarks on the extended care facility. For all practical purposes the extended care facility is a Medicare approved nursing home to which hospital patients who still need medically supervised, skilled nursing and related services, such as physiotherapy, in an institutional setting, are admitted for further care. It is also designed to care for the post-acute hospital patient convalescing or being rehabilitated, and third and finally is intended to care for the severely ill with no hope of recovery such as terminal cancer patients.

Patients may be transferred after at least three days in a Medicare hospital to a Medicare approved extended facility for the first twenty days at Government expense, and for an additional eighty days at a cost to the patient of five dollars per day, so long as they qualify for extended care as above described, but only so long. Should they fail to meet the qualification for this type of care they descend to the next level of patient care known in Governmentese as Custodial Care.

Custodial care is not underwritten by Medicare and its expense must be borne by the patient. Custodial care is defined as that type of care which is designed to assist an individual to meet his activities of daily living, i.e., services which constitute personal care such as help in walking, getting in and out of bed, assistance in bathing, etc., which does not entail or require continued attention of trained medical or paramedical personnel.

Thus, the physician cannot even admit a patient to a hospital unless he needs hospital care, and if he didn't need hospital care, he would not be eligible for Medicare in a nursing home even though he was hospitalized for three days.

Thus custodial care of a patient, which is not covered by Medicare funds, is determined by the level of care needed for his reasonable maintenance rather than upon such considerations as diagnosis, severity of the disease or degree of functional limitation.

Each physician is expected to exercise his professional judgment on the one hand to see that his patient avails himself of everything helpful that the benevolent government has to offer, such as rehabilitative and occupational and other ancillary services, and on the other, to prevent abuse of these same facilities by patients who expect a "free ride" for twenty days and a "subsidized ride" for another eighty days for conditions that under the law are not allowable.

Each nursing home is required by law to have a Utilization Committee consisting of at least two physicians and one or more professional ancillary personnel, which is charged to review Medicare patients at least once monthly. They may recommend cessation of certification for further care, but rely on the judgment of the attending physician.

We as physicians thus have a clear cut duty to our patients and to our Government, no matter how onerous it may be, to acquaint ourselves with regulations as set down for us and try to set a course which will be fair to all parties concerned. May I commend a new publication issued by the AMA entitled, "The Extended Care Facility, a Handbook for the Medical Society," which is available at 90 cents a copy from AMA Order Handling Department, 535 North Dearborn Street, Chicago, Illinois 60610.

We are in for more regulation, more red tape, more control, as we warned, of the socialized camel's nose in the tent of medicine. But, I am afraid we must make the tent larger so that we can live comfortably with the camel. Who would have thought that Americans would be compelled to live that way!

John P. Lynch, M.D.
Chairman, Medicare Committee
Richmond Academy of Medicine

1000 West Grace Street
Richmond, Virginia

Hearing Aids and Hearing Aid Dealers

CARY N. MOON, JR., M.D.
Charlottesville, Virginia

Before the hard of hearing patient purchases a hearing aid he should have a thorough work-up by an otolaryngologist. The purchase of hearing aids direct from the salesman often results in disappointment and waste of money. Recommendations for the control of the advertising and selling of hearing aids is presented.

SEVERAL OF US in Otology have expressed concern about our hard of hearing patients who have purchased one or more hearing aids without previous medical examinations, hearing aids which frequently were not audiologically indicated or were improperly fitted. The patient with the sensori-neural hearing loss coming on in later life, with poor discrimination ability and noise intolerance, seems to be particularly vulnerable to the sales pressure practices of hearing aid salesmen.

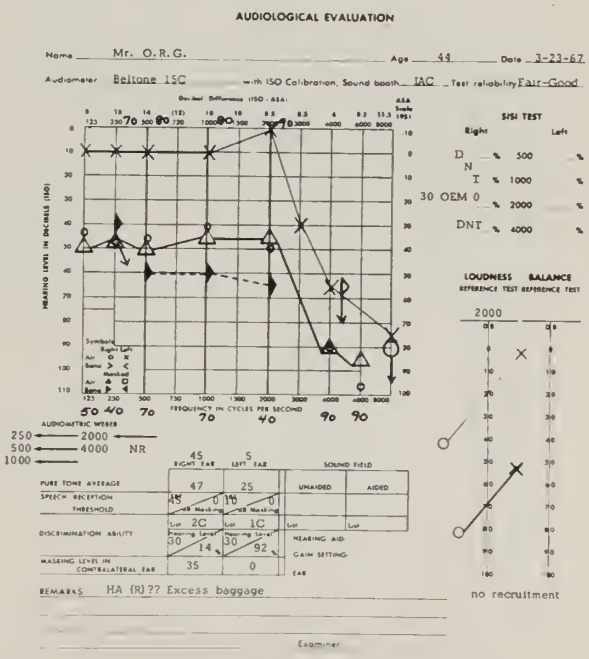
This report will briefly review this subject. Four case reports will be given as examples of inappropriate sales of hearing aids. There will be a discussion of advertising, prices of hearing aids, and the hearing aid dealers. The ear doctors' responsibilities to the hard of hearing patient and to the hearing aid dealer will be outlined. In the conclusion, some recommendations that appear to be indicated will be suggested.

Presented at the annual meeting of The Virginia Society of Ophthalmology and Otolaryngology, Hot Springs, Virginia, April, 1967, and The Medical Society of Virginia, Arlington, October 1967.

From the Department of Otolaryngology, School of Medicine, University of Virginia, Charlottesville, Virginia.

Case Reports

Case No. 1. This is an excellent example of the fallacy of hearing aid selection based on the information provided by the pure tone audiogram. It would appear that this man would be able to achieve relatively balanced hearing with the use of a mild-to-moderate gain hearing aid in the poorer ear. However, when you consider his discrimination ability in the impaired ear, it is apparent that the hearing aid in that ear

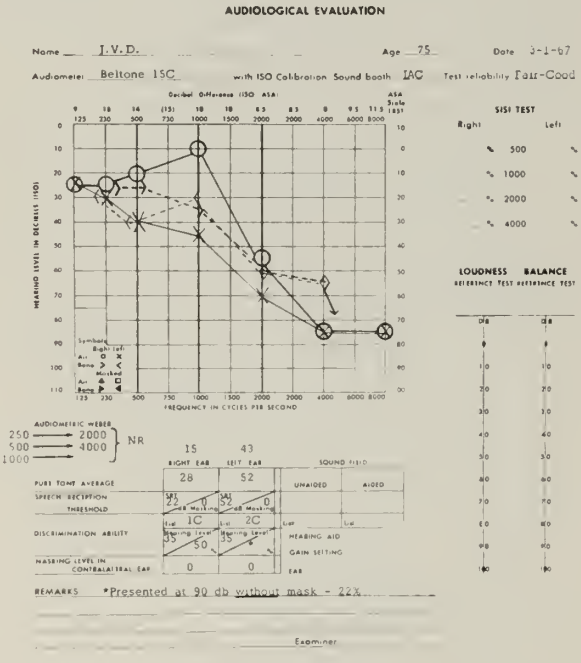


Audiogram No. 1

is not indicated as the aid will present such distorted information that there is a possibility that it would adversely affect the hearing reception in the better ear. Furthermore, it is noted that the audiological

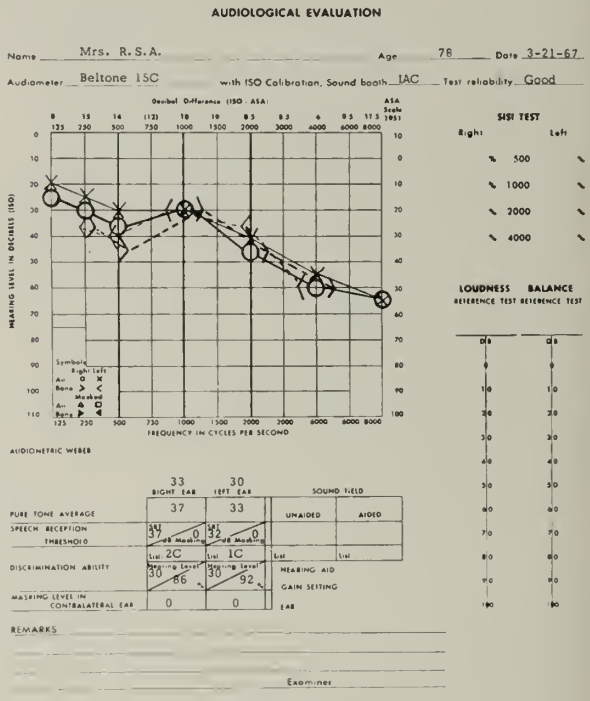
findings are suggestive of an eighth nerve lesion, possibly an acoustic neuroma. He had purchased and attempted to use an ear level aid prior to seeking an otological examination.

Case No. 2. When first examined, this patient was wearing a binaural eyeglass hearing aid arrangement. He was somewhat satisfied with his aids. He was a self-referral to the dealer, and his chief reason for seeking further attention was his concern for the expense of the aids and their upkeep. He stated that it cost him $54.00 to return the aids to the factory, and that there was something wrong continually. It was our opinion that this man should not be wearing an aid in the right ear; it was too good for amplification. We would not have recommended a hearing aid for this patient had he been seen prior to this $650.00 investment.



Audiogram No. 2

Case No. 3. The hearing aid door-to-door salesman wanted to sell this woman a $379.00 hearing aid, in-the-ear-model, for her right ear. Fortunately, she was home alone at the time, and she told the salesman that she could not act without consulting her children on a matter of such expense. She was informed that she had a 75 per cent hearing loss at 10 feet and, furthermore, he told her that any loss greater than 10 decibels was subnormal. We advised her not to purchase a hearing aid, as we felt her speech reception thresholds were still too good to require amplification.

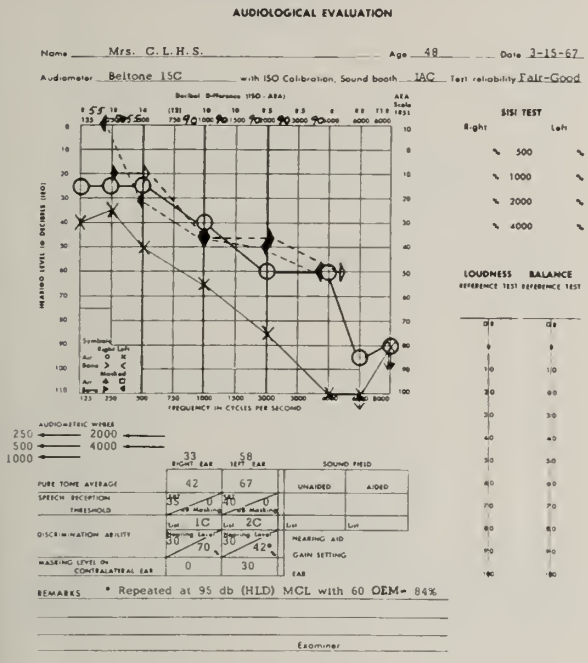


Audiogram No. 3

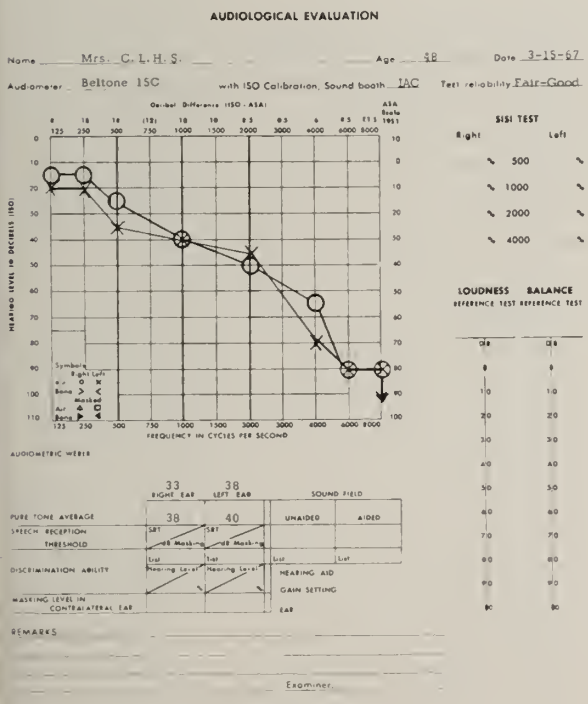
Case No. 4. This 48-year-old woman had an eyeglass hearing aid fitted for the right ear six months prior to this evaluation. She had a history of chronic purulent drainage from both ears, of many years duration, with an infectious eczematoid dermatitis involving the ear canal and the auricle on each side. She was sold a "non-allergic ear mold" which, according to the salesman, could be used in the presence of drainage. She complained that her ear mold "popped out" and that she could only wear her aid for a few hours at a time. After cleaning the canals, there was serviceable hearing in the right ear; and after a tympanoplasty, there was serviceable hearing in the left ear.

In our files, there are other cases of aids being fitted and sold when the hearing loss was due to middle ear fluid, ear canal wax,

and other obstructive causes. Fortunately, such cases do not represent the majority, but probably only 10 or 15 per cent of the



Audiogram No. 4



Audiogram No. 5

total. There is probably another 10 or 15 per cent of borderline cases that properly should have been given a prolonged trial period rather than an irrevocable sale of a hearing aid. These particular patients are usually so unhappy with the instrument that they do not even bother to bring it with them for their otological-audiological evaluation. Approximately 80 per cent of those people who have purchased hearing aids have done so without benefit of an ear doctor's evaluation. Many of them have bought several aids on the advice of hearing aid dealers that the newer or different instrument would outperform the previous one. The extreme example of this was one man who had nine hearing aids, and stated that he had spent in excess of $3,000.00 on these instruments over a period of some five years. After otological-audiological evaluation, it was our opinion that amplification would do him no good.

Advertising

Hearing aid advertisements are frequently misleading and suggest sensational relief of all kinds of deafness. They inquire, "Do you want to hear again?" Most of these advertisements request that you complete the form, giving your name and address; reassuring you that there is absolutely no obligation, and some promise that no one will call.

There must be strict regulations for all advertisements concerning hearing aids. These advertisements should be restricted to the name of the dealer, the office address, and the name of the particular aids for sale. It should be stipulated that it is unethical to use terms with medical suggestions such as clinic, hearing center, or audiologist. The words, Hearing Aid Dealer, should be clearly displayed.

The Cost of Hearing Aids

The high price of hearing aids is explained on the basis of the relatively low volume of

sales, for instance, as compared to the high volume of sales of television sets. The high price is a reflection of the method of distribution and supposedly covers guarantee periods and periods of service without fee. There is information that the retail price exceeds the wholesale price by 150 per cent to 300 per cent. Consumers Reports, with reference to hearing aids, January 1966—Vol. XXXI, No. I, states, "There is a strong similarity among them all in general design and the kind of parts used, nor did the assembly techniques of the expensive aids exhibit an especially high precision to justify their high cost."

The price of hearing aids is generally above $300.00 an instrument, but it is apparent that there is some very questionable discrepancy when Zenith has a body type, high amplification instrument for $75.00 and Sears has a moderate amplification, ear level model for $83.00. Both of these relatively low cost instruments were called a "Best Buy" in the Consumers Reports on Hearing Aids.

The Hearing Aid Dealer

The hearing aid salesmen can generally be classified into two groups. One is composed of honest, highly motivated individuals with a sincere desire to be of assistance in the rehabilitation of the hearing-handicapped individual. The other group is composed of supersalesmen with but one aim: to make the sale.

The Hearing Aid Dealers Association is a very worth while organization of many ethical dealers whose purpose is to improve their service to the hard of hearing and to improve the public image of the hearing aid dealer. Their folder states that they were organized to "establish and improve standards of performance in the fitting and selling of hearing aids by its members."

VIRGINIA HEARING AID
DEALERS' ASSOCIATION
INCORPORATED

MEMBERSHIP
DIRECTORY



Member
National Hearing Aid Society

Fig. 1

The Hearing Aid Dealers Association has an ethics committee. They inform me that they can reprimand and punish wrongdoers within their own organization.

The ideal hearing aid dealer must have a basic knowledge of hearing testing methods, must have the fundamental up-dated well-calibrated equipment for testing hearing, must know his hearing aids and the technique of their repair, and he must have available accessory supplies. He must insist on a preliminary otological and audiological examination prior to hearing and fitting and hearing aid sale.

The hearing aid dealer should always be aware of his responsibility to the hearing-handicapped, and above all must be honest. The income-producing aspects of each potential sale have to be secondary to service.

He should properly be a member of the rehabilitation team and avoid the salesman-customer relationship. Properly, the hearing aid dealer can continue to seek out the hard of hearing individual who needs help, but then he must insist on an otological-audiological work-up to determine the diagnosis and individual suitability for amplification.

We would suggest that the hearing aid dealer diversify and expand his business to include items other than hearing aids: items such as headset earphones, stereophonic and monophonic attachments for radio, record players, and television, desk amplifiers, ear trumpet hearing aids, and custom-built ear plugs to protect ears from excessive noise or from contamination by water.

There is a real need in Virginia for local sales of audiometers and audiological testing equipment and the calibration and repair of this equipment. There are probably a dozen or more otological offices and six hearing clinics within the State that would welcome this local sales and service.

I believe that the hearing aid dealer meeting these qualifications and requirements, and thus facing his responsibilities to the hard of hearing, can become an essential part of the over-all rehabilitation of the hearing-handicapped.

The Otolaryngologist

We, otolaryngologists, have displayed a certain lack of interest in the hard of hearing patient with sensori-neural hearing loss. We have been guilty of dismissing these patients without proper counseling and without proper follow-up attention. The doctor has been too inclined to assume no responsibility for the hearing aid selection and no interest in the results of amplification. There has been little or no communication between the ear doctor and the hearing aid salesman.

It is our duty to give all hard of hearing patients a serious, thorough examination and evaluation. This necessarily must include pure tone audiometry, bone-air thresholds, speech reception and speech discrimination testing, and more specific diagnostic studies when indicated. Preferably, these studies are done by a qualified audiologist in a sound-treated area. The audiological work-up has to include a routine ear, nose and throat examination, the required history, and the tuning fork tests. The tuning fork tests are considered by some to be antiquated; they are rough determinations, but do unite the otological examination to the audiological examination, confirming the accuracy or inaccuracy of the pure tone hearing testing.

It is the very important responsibility of the otologist to discuss with the patient the diagnosis, what can be expected in the future, and the possible avenues of relief from this hearing loss. Now—the specialist has to become a true physician. He has to be sympathetic, reassuring, and interested. Hearing aids should be discussed, and the patient must be advised what to expect and what not to expect. The advantages of

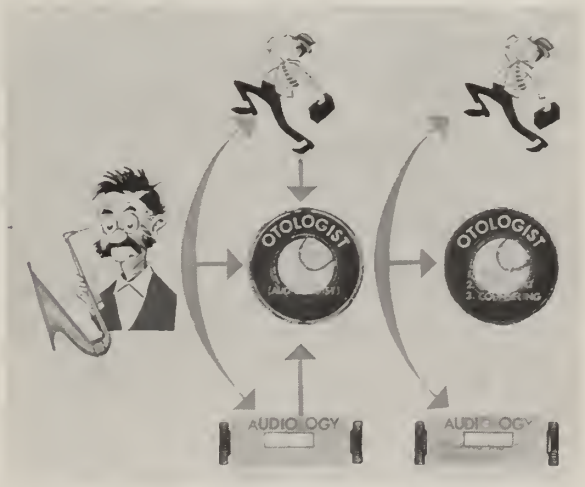


Fig. 2

amplification should be emphasized, but also the patient should be informed about the problems of discrimination. All too frequently, the individual buying a hearing aid expects normal hearing, and is quickly disappointed. This may result in a discarded instrument. However, if he is properly

counseled, he is better prepared to recognize the advantages of amplified sound, and can more readily accept the disadvantages with little or no complaint. The ear doctor must accept his share of the responsibility, along with the hearing aid salesman, in the rehabilitation of the patient requiring a hearing aid. A closer association of the doctor and the hearing aid dealer, and also the audiologist, can only result in a better-treated and happier hard of hearing patient.

Recommendations

No. 1. We endorse the basic requirement of a complete hearing evaluation.

(a) An ear, nose and throat examination and history by an otolaryngologist.
(b) An audiological evaluation by an audiologist.

No. 2. We endorse the Virginia Hearing Aid Dealers Association, Incorporated. We commend them for their efforts, and approve of their organizational aims. It is recommended that we support the Virginia Hearing Aid Dealers Association.

No. 3. We recommend legislation for examination and licensing with a certificate of registration, in order to become a hearing aid salesman in the State of Virginia.

No. 4. It is recommended that there be a committee to review the patients', the hearing aid dealers', or the physicians' complaints of errors in the selling of hearing aids; that this committee make recommendations as to the correction of the wrong, if one does exist, and reprimand wrongdoers directly, requesting that the error be corrected; and that if this does not settle the problem, then a report should be made to the Hearing Aid Dealers Association and to the agency company involved.

No. 5. We endorse and approve of the Virginia Retail Sales and Use Tax Rules and Regulations No. 1-65, which states that the sales tax does not apply to the hearing aid sold on prescription or work orders of a licensed physician. However, the wording in this section suggests that optometrists, opticians, and dentists can sell hearing aids without tax, which seems to discriminate against the hearing aid dealer who does not happen to be an optometrist, optician, or dentist. The tax exempt aspect of a hearing aid prescribed by a physician would seem to encourage the patient to seek a medical examination before purchasing a hearing aid. A paragraph should be added to Section 1-65, clearly stating that a licensed physician or an audiologist, at the request of a physician, can give a work order for a hearing aid which will be tax exempt.

Conclusion

Amplification through an individually-fitted hearing aid has to be a type of medical treatment requiring the diagnosis and the prescription of a medical doctor. The hearing aid salesman system of finding the hearing-handicapped, diagnosing the deafness, prescribing the cure, and selling the aid without medical consultation cannot be permitted to continue.

The refined diagnostic procedures of the otologists and the audiologists are essential to locate and identify the cause of deafness. Today, we have available a battery of hearing tests which occasionally suggest the diagnosis of an acoustic neuroma, even when this neoplasm is still within the internal auditory canal and when it is possible to surgically remove this lesion with reduced risk and reduced sequelae. All patients with sensori-neural hearing losses, in particular those with unilateral losses of this type, require expert audiological testing. Most patients with conduction hearing losses can obtain relief by medical or surgical treatment.

We invite the hearing aid dealers to join us in the over-all treatment and rehabilitation of the patient with a hearing loss.

1000 East High Street
Charlottesville, Virginia 22901

Hospital Experience with Acute Myocardial Infarction

DONALD R. HOLSINGER, M.D.
Martinsville, Virginia

The advent of the intensive coronary care unit in many hospitals may have started a new era in the management of acute myocardial infarction.

A BREAKTHROUGH in coronary heart disease has been accomplished through the use of the intensive coronary care unit and its monitoring and treatment methods. There is an air of dynamic change in the care of the "coronary" victim and the new techniques being developed are applicable to practically all general hospitals, regardless of size. Continual monitoring, cardioversion of arrhythmias, and intensive nursing care of the coronary patient, if not already in use, will soon be commonplace in the management of the victim of acute myocardial infarction. Investigative programs are yielding a better perspective on the treatment of cardiac shock and congestive heart failure and hopefully these programs will present new techniques for the handling of these complications of myocardial infarction.

Evaluation of these new treatment methods is of critical importance if they are to find their proper place in the treatment program of coronary heart disease.

In view of this need for careful evaluation of the rapidly changing parameters in the care of acute myocardial infarction, it was felt that a study of the results of recent, but now perhaps obsolete, management methods was needed. The results of this will be used for comparison with later studies incorporating the newer techniques. Thus, the ultimate value of the new techniques will be judged.

From the Department of Medicine, Martinsville General Hospital, Martinsville.

Method

Medical records from January 1, 1963, through December 31, 1964, at Martinsville General Hospital, of patients diagnosed as acute myocardial infarction were reviewed. During these years routine monitoring was not used and intensive care in a coronary unit was not available. Those records which did not reveal objective evidence of myocardial infarction were discarded. During this period there were 10,975 admissions and of these 114 had documented acute myocardial infarction. These records were reviewed with particular emphasis on diagnostic and management methods and electrocardiographic factors in the diagnosis of complications and their treatment.

Results

Of the 114 cases of acute myocardial infarction there were 31 deaths (27 per cent) during hospitalization. There were 93 males and 21 females, with a male to female ratio of 4.4:1. Among the males there were 25 deaths (27 per cent) and in the females, 6 deaths (29 per cent). The age to incidence relationship and the corresponding deaths is shown in Figure 1. The peak incidence occurred in the fifties and is

similar to the study of Master et al.[1] The death rate was highest in the seventies when 43 per cent of the patients died.

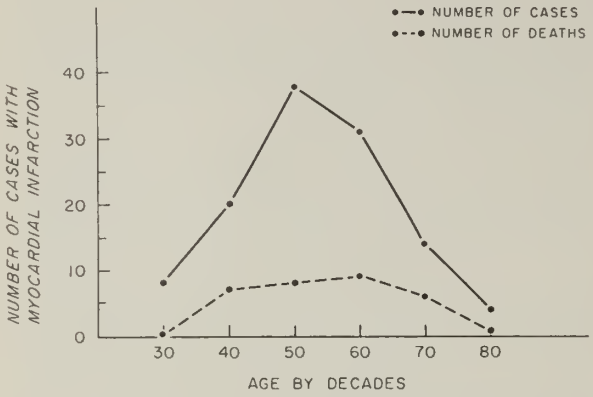


Fig. 1. Incidence by age of patients with acute myocardial infarction and the incidence of corresponding deaths.

The number of deaths on consecutive hospital days is shown in Figure 2. The highest mortality, 10.5 per cent, occurred on the first hospital day. The mortality for the first seven days including the first day deaths was 20 per cent, which accounted for approximately four-fifths of all the hospital deaths. For the remaining hospitalization after the first week there was an additional mortality of only 7 per cent.

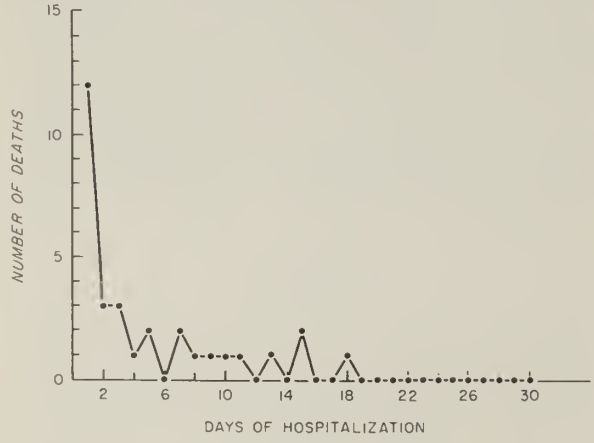


Fig. 2. Number of deaths by day of hospitalization.

Diabetes was confirmed in only eight patients; this is probably lower than the true incidence and may reveal that "prediabetes" was not detected as often as possible. Hypertension was recorded in 19 patients and a previous myocardial infarction in 20 patients. Of the 28 patients having a blood cholesterol determination, 14 (50 per cent) had values greater than 250 mg. per 100 cc., but the number in this group is too small to be statistically significant.

Table I gives the electrocardiographic location of the infarctions and their incidence as well as the associated mortality of each.

TABLE I

Electrocardiographic Location of the Infarction and the Associated Mortality

Location	Number	Deaths
Anterior	50	15
Diaphragmatic	44	7
Anterior and diaphragmatic	5	3
Sub-endocardial	6	0
Not localized	9	6

There were 55 major complications in the 114 cases, and as expected more than one complication occurred in some cases. Arrhythmia and congestive heart failure were the most frequent complications and carried sizable associated mortality rates. Cardiac shock and pulmonary embolus were less frequent but were more often fatal. Two of the infarctions occurred postoperatively and both were fatal.

Electrocardiographic diagnosis of arrhythmia was made in 24 patients or in only 21 per cent of the patients. Table 2 shows the incidence of the various arrhythmias and the frequency of deaths in each. In the 24 patients who had detected arrhythmias there were 11 deaths giving a 46 per cent mortality in this group.

Left bundle branch block was present in four patients and right bundle branch block in two.

Congestive heart failure occurred in 21 patients and was associated with a fatal outcome in 10 cases (46 per cent). Three of 5 patients who had diagnosed cardiac shock died. Considerably more than these 5 patients received vasopressor drugs; possibly this reflects the use of pressor agents in the treament of other complications in a terminally ill patient.

Seventy-six of the 114 patients received anticoagulation therapy using heparin, warfarin, or both. Nineteen received digitalis; fifteen received vasopressor drugs and six received anti-arrhythmic drugs. Of these various treatment modalities only those patients requiring vasopressor drugs exhibited a higher than average mortality rate

TABLE II

COMPLICATIONS IN 114 CASES OF ACUTE MYOCARDIAL INFARCTION AND THE ASSOCIATED MORTALITY

COMPLICATION	Number	Deaths	% Deaths
Cardiac shock	5	3	60
Arrhythmia	24	11	46
Congestive failure	21	10	46
Associated "stroke"	4	0	0
Pulmonary embolus	1	1	100

where 12 of 15 patients so treated died. This high mortality vividly reflects the inadequacy of the treatment of cardiac shock by the methods now available.

TABLE III

FREQUENCY OF ARRHYTHMIAS AND THE ASSOCIATED MORTALITY

TYPE OF ARRHYTHMIA	Number	Deaths
Frequent premature ventricular beats	1	0
Atrio-ventricular block—		
1st degree	3	0
2nd degree	3	2
3rd degree	7	3
Atrial flutter	1	1
Atrial fibrillation	3	2
Ventricular tachycardia	3	1
Ventricular fibrillation	3	2

Comments

The overall mortality of 27 per cent is higher than the previously reported series of Bland and White[2] who reported 19 per cent mortality in 200 cases and Master et al.[3] who reported 16.5 per cent mortality in 267 cases. It is felt that the series of Bland and White, and Master reveal a lower mortality probably because these patients had passed the most dangerous early phase of their illness before they were seen by these consultants. Ball et al.[4] in 1955 reported a 26 per cent mortality in 342 patients in the first four weeks of their illness; the method of selection of the cases and the results of Ball compare closely with the present study.

Two areas of possible improvement in the care of myocardial infarction seem apparent from this study. First, more frequent use of anticoagulation therapy; only two-thirds of the patients in this study received anticoagulation therapy. Most studies reported in this country and abroad shows improved mortality in the acute phase with the use of anticoagulant therapy in the short term care of myocardial infarction. This should not be confused with the debatable use of long term anticoagulant therapy where the results are less clearly defined. Secondly, the improvement in the detection of arrhythmias would certainly be beneficial. In this series of 114 patients, arrhythmias were detected in only 21 per cent of the cases. While in other reported series using continuous monitoring for 72 hours to 12 days there are reported incidences of 60 to 90 per cent of arrhythmias detected during these periods.[5,6,7,8,9] However, the high mortality rate of 46 per cent of detected arrhythmias in the present series is much higher than the death rate from arrhythmias where arrhythmias are detected more frequently with the use of continual electrical monitoring. Probably the high death rate from arrhythmia in the present study is due to the persistence of the significant arrhythmias demanding or resulting in their discovery by physical examination or conventional electrocardiography while transient arrhythmias were not detected.

Seventy-five to eighty-five per cent of the deaths from myocardial infarction occur within the first week of the attack and the vast majority of these deaths result from cardiac arrhythmias, cardiac shock, or congestive failure, alone or in combination. It appears that prompt and effective treatment will significantly reduce this mortality, especially if potentially lethal cardiac arrhythmias are promptly detected and treated utilizing modern techniques. This requires continual attention and immediate action.

It would appear that the care of all acute myocardial infarction patients would be accomplished best in an intensive care unit with continuous electrical monitoring and equipment necessary for resuscitation close at hand. The early detection of cardiac arrhythmias and an organized approach to the treatment and resuscitation of patients suffering from these complications would then be possible. Hopefully, this would result in a lowering of mortality from coronary heart disease.

Careful clinical evaluation of these new techniques is an essential component of these studies. Through this study it is hoped that a baseline has been established in this hospital against which the new techniques and methods which are being implemented and planned for the future can be evaluated.

Summary

The two year experience in the care of the acute myocardial infarction at Martinsville General Hospital for 1963-64 has been reviewed. One hundred and fourteen cases were reported with a 27 per cent mortality. The complications and their management were studied. Hopefully, this study will serve as a baseline for later studies incorporating new techniques or treatment of coronary heart disease.

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15 Cleveland Avenue
Martinsville, Virginia 24112

Study of Patients with Lymphosarcoma

The cooperation of physicians is requested in the referral of patients with lymphosarcoma for a study being conducted by the National Cancer Institute at the Clinical Center, National Institutes of Health, Bethesda, Maryland.

Patients with biopsy proven disease who have received no treatment are eligible for admission as inpatients for diagnostic evaluation and treatment as indicated. Upon completion of their studies, patients will be returned to the care of the referring physician, who will receive a summary of findings.

Physicians interested in having their patients considered for admission to this study may write or telephone: Robert B. Howe, M.D., Clinical Center, Room 4-B-09, National Institutes of Health, Bethesda, Maryland 20014. Telephone: 656-4000, Ext. 62031 (Area Code 301)

Gastrostomy in Infants

Indications and Management

GEORGE A. HARKINS, M.D.
Norfolk, Virginia

Temporary gastrostomy in infants is a helpful procedure and probably should be used more frequently.

DURING the postoperative period most infants are in precarious balance, apt to improve under optimum conditions. Among others, these optimum conditions include a great deal of rest, the ability to clear secretions, the avoidance of respiratory complications, and proper nutrition. The surgeon must provide these optimum conditions in so far as possible. Generally this requires reliable gastric decompression, accurate knowledge of the postoperative functional state of the gastrointestinal tract and adequate nutrition. The temporary gastrostomy often is helpful in meeting these needs.

Respiratory complications, principally caused by distention or vomiting, must be prevented by adequate gastrointestinal decompression. The nasogastric tube, which serves well in toddlers and older children is not reliable in infants. Its small size, generally 8 or 10 French, predisposes to obstruction. Unfortunately, the function of the lumen is not related to the size of the patient, but to the viscosity of the fluid. Secretions from a small stomach need as large a tube as those coming from a large stomach. In addition, the nasogastric tube, even though small, will come to occupy a relatively large area in the pharynx and prohibit effective swallowing. This in itself causes repeated aspiration. Consequently, the Levin tube in infants not only does not provide continuous decompression, but actually contributes to pulmonary complications.

Gastrostomy is ideally suited for gastric decompression in the infant. Its size is not limited and generally a number 14 to 18 French is selected. Drainage is reliable regardless of the viscosity of the secretions.

This indication, the provision of constant reliable decompression, is the principal indication for the use of gastrostomy in infants. Once in place, however, it has adjunctive uses which are considerable. The most important is accurate determination of gastrointestinal function. As in any other type of decompression the time to feed and quantity acceptable can be judged by the color and amount of drainage through the decompression tube. In addition, by aspiration through the gastrostomy prior to a feed, one can judge whether the feeding should be increased or decreased.

Its use as a feeding mechanism is obvious. The ease and safety of gastrostomy feeding can be decisive in the care of the weak or debilitated infant. In the extreme premature (under one kilogram) the safety of gastrostomy as compared to oral feeding or gavage is indication enough for temporary gastrostomy.[1]

Table I is a list of the conditions for

Presented at the annual meeting of the Virginia Surgical Society, Staunton, April 28, 1967.

Table I

Diagnoses in Infants Under Six Months of Age in Whom Temporary Gastrostomy Was Performed

Esophageal Atresia	8
Spontaneous Perforation of Stomach	2
Duodenal Atresia	1
Ileal Atresia	1
Meconium Ileus	1
Pierre-Robin Syndrome	1
Necrotizing Enterocolitis	1
Ruptured Omphalocele	1
Peritonitis Secondary to Pylormyotomy	1
Biliary Atresia (Correctable)	1
Hiatus Hernia	1
Malrotation	1
Total:	20

which we have used gastrostomy in infants under six months of age during the past three years. In summarizing specific indications, we would recommend the temporary gastrostomy in infants for the following conditions: 1. All cases of esophageal atresia and tracheo-esophageal fistula; 2. Any case in which gastric decompression is apt to be desirable for more than 24 hours; 3. The premature or distressed baby undergoing surgery; 4. Respiratory complications requiring performance of tracheostomy; 5. Extreme prematurity; 6. Occasional special situations, such as extreme feeding problems or perforated esophagus.

This list ignores the use of gastostomy in older children in whom it is of benefit in a variety of problems, including chemical burns of the esophagus, hiatus herniorrhaphy, coloesophagoplasty, and tetanus.

The recommendation for the use of temporary gastrostomy presumes that the procedure is safe. This is correct provided certain precautions are taken. Local anesthesia is preferred. Operative time is brief, and, even so, provision is made to maintain body temperature during surgery. The Stamm gastrostomy has proven to be the safest technique in infants. A site high on the anterior surface of the stomach, preferably away from the greater curvature, is selected. Non-absorbable suture is used, both for the purse strings holding the gastrostomy into the stomach and those sutures anchoring the stomach to the under-surface of the abdominal wall. A mushroom catheter or similar premolded soft tube is selected.

The complication rate is low and no significant complications need be anticipated.[2] In the 20 cases in this series there were no major complications. The only minor complication was a leak around the gastrostomy, easily corrected by tightening the dressing.

A potential hazard is premature or accidental removal of the gastrostomy.[3] The Foley catheter has proven to be hazardous in infants because accidental deflation of the balloon may allow premature removal of the tube. Disruption of the seal between the stomach and the abdominal wall and secondary peritonitis may then occur. It is recommended that the gastrostomy be left in for a minimum of 10 days even when no longer in use. Should the gastrostomy inadvertently come out in the first several days, the baby should be returned to the operating room for formal surgical replacement. This complication has not occurred in this series of patients.

Management of the gastrostomy is largely a nursing problem. However, many nurses have little understanding of this tube and its care. A firm dressing should be applied to the base of the gastrostomy and abdominal wall. Dressing change is generally undesirable during the first week. Restraints to the hands are seldom necessary. Suction is neither necessary nor desirable. The distal end of the tube should be placed into the open mouth of a test tube or tied into a distensible rubber glove.

When it appears that feedings can be started, the tube is elevated 8 to 10 cm. above the abdominal wall and left open for six to 12 hours prior to commencing gastrostomy feeding. If significant backflow through the tube has not taken place, a slow drip is started in small amounts and gradually increased. The feeding setup includes a Murphy bulb with an air vent which is kept no higher than 20 cm. above the abdomen. If distal obstruction or reverse peristalsis occur, the gastrostomy then func-

tions as a safety valve preventing vomiting. Aspiration of the stomach through the gastrostomy prior to each feeding yields further valuable information.

The gastrostomy is a handy means of removing air from the stomach and avoids the necessity of burping. This reduces the need for handling the sick infant. Feedings should be dripped in slowly, approximating the time that would be required to feed the same amount orally.

When improvement has occurred and the patient has demonstrated steady weight gain, the gastrostomy may be abandoned in favor of oral feeding. However, there are many occasions when oral feeds are possible and yet not desirable. Switching from gastrostomy is not done as long as there is question of weakness or debility of the infant. Once on oral feeding, the tube still is useful as a substitute for burping.

Following permanent clamping of the gastrostomy tube and satisfactory weight gain on oral feedings, the gastrostomy is removed and a small pressure dressing applied. The wound will generally seal within a few hours. We have had no instance where the gastrocutaneous fistula has not healed rapidly in infants. In older children in whom the gastrostomy has been in place for months or years, the fistula may become lined by gastric mucosa and require formal operative closure.

Our only regret regarding temporary gastrostomy in infants have been in those cases where it might have been of value and we have neglected to use it. We are continuously liberalizing our indications for its use and recommend it as a safe and desirable adjunct in the care of these tiny patients.

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Study of Patients with Bilateral Cryptorchidism

The cooperation of physicians is requested in the referral of patients with bilateral cryptorchidism for studies being conducted by the Surgery and Endocrinology Branches of the National Cancer Institute at the Clinical Center, National Institutes of Health, Bethesda, Maryland.

Needed for these studies are patients of all ages with bilateral cryptorchidism. Patients in whom exploration is contemplated or in whom exploration has already failed to reveal gonads are of particular interest. Upon completion of their studies, patients will be returned to the care of the referring physician who will receive a summary of findings.

Physicians interested in having their patients considered for admission to these studies may write or telephone: Elwin E. Fraley, M.D., Clinical Center, Room 10-N-119, National Institutes of Health, Bethesda, Maryland 20014, Telephone: 656-4000, Ext. 63351 (Area Code 301), or Marvin A. Kirschner, M.D., Clinical Center, Room 12-N-206, National Institutes of Health, Bethesda, Maryland 20014. Telephone: 656-4000, Ext. 63079 (Area Code 301)

The Trisomy 17/18 Syndrome

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The radiographic and somatic findings of the 17/18 trisomy syndrome constitute a diagnostic entity which is being recognized with increasing frequency. This report constitutes a presentation of the characteristic feature of this syndrome based on a review of the literature and our own experience with two cases. Awareness of the features of this syndrome are important to the radiologist and pediatrician since evaluation of this genetic condition is not available in many communities.

THE RADIOGRAPHIC and somatic findings of the 17/18 trisomy syndrome constitute a diagnostic entity which is being recognized with increasing frequency. This report constitutes a presentation of the characteristic feature of this syndrome based on a review of the literature and our own experience with two cases. Awareness of the features of this syndrome are important to the radiologist and pediatrician since evaluation of this genetic condition is not available in many communities.

The 17/18 trisomy syndrome, which is characterized by an additional chromosome in the 17/18 group, was first recognized by Edwards, et al., in 1960 by growth cultures from skin and muscle—both contained 47 chromosomes.[3] One chromosome exists in triplicate resembling both pairs 17 and 18 of the normal complement with respect to size and position of the centromere. In mongolism (Down's Syndrome), the extra chromosome is due to triplication (trisomy) of chromosome 21.[4] There have been reports of 115 cases of trisomy 17/18 syndrome in the literature.[1,2] At birth, these patients have multiple anomalies and approximately 90% die by age one year.[7] They are usually female, mentally retarded, and are progeny of older parents. There is apparently no increased incidence within the families of these patients.

Case I. D. H., a white female, was born March 1963. She was the third child of parents whose ages are unknown. At birth, the infant weighed five pounds, seven ounces. The pregnancy was apparently uncomplicated. At birth, she was thought to have Down's Syndrome. At age one month, she was transferred to the University of Virginia Hospital where the following abnormalities were noted: occipital prominence, micrognathia, syndactyly of the second and third toes bilaterally, rocker bottom feet and low-set malformed ears. The palate had a high arch, but no fissure cleft. Simian lines in both palms were noted, as well as flexion deformities of the fingers with ulnar deviation. A grade 3 of 6 systolic murmur was heard over the entire precordium. The chest had a shield-like configuration.

Radiographic skeletal studies were performed at age one month. These revealed hypoplasia of the mandible and relative en-

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largement of the tongue. The posterior fossa of the skull appeared increased in the anteroposterior diameter. The ribs appeared gracile with hypoplasia of the manubrium and no visible sternal ossification centers. The heart was markedly enlarged with increased pulmonary vascular markings (Fig. 1). The pelvis was small. The hands showed slight ulnar deviation with the third finger overlapping the second. A V-shaped gap was noted between the second and third fingers when the hand was straightened (Fig. 2). An equinovarus deformity of both feet was noted with non-ossification of the middle phalanges of the third, fourth and fifth toes. The first (great) toe was smaller than the second bilaterally with a V-shaped gap between the two (Fig. 3).



Fig. 1. Case I. Chest roentgenographs demonstrate cardiomegaly with increased pulmonary vascular markings. (Subsequent cardiac catheterization demonstrated a large ventricular septal defect). The anteroposterior diameter of the chest is increased, and the ribs are thin. Note the absence of discernable sternal ossification centers.

Blood cultures, blood chemistries and urinalysis were all within normal limits. Growth and development were retarded during a one month hospital course. Chromosomal analysis was done from peripheral blood leucocytes by a modification of the Moorehead method. The cells contained 47 chromosomes, but it was not possible to determine whether the extra chromosome was 17 or 18 (Fig. 4).

Clinical Course: Patient was discharged and died at age 12 weeks; no autopsy was obtained.



Fig. 2. Case I. Demonstrates V-shaped gap between second and third fingers.

Case II. M. D., a white female, was born January 1962 weighing four pounds, 11 ounces. The age of the parents and length

of gestation are unknown. She was admitted to the University of Virginia Hospital with a diagnosis of congestive heart failure. It was noted at birth that the ears were flattened against the head and the hands flexed with slight contracture which was difficult to overcome by the examining pediatrician.

Physical examination demonstrated a small wizened face with low-set "elf-like" ears. Flexion at the metacarpophalangeal joints was noted with hypertonicity of hands and clubbed feet. A grade 3 of 6 systolic murmur was heard. The mandible was receded with a small mouth. The external genitalia were normal.

Blood chemistries, urinalysis, stool and cerebrospinal fluid were all normal. Electroencephalogram was normal. Electrocardiogram showed left ventricular overload. Except for transient pneumonitis and episodes of congestive heart failure, little change was noted during a nine week hospital course.



Fig. 3. Case I. Equinovarus deformity of the forefoot. There are no ossification centers for the middle phalanges of the third, fourth and fifth toes. The terminal phalanges are small. The first toe is short and blunt with a V-shaped gap between the first and second.

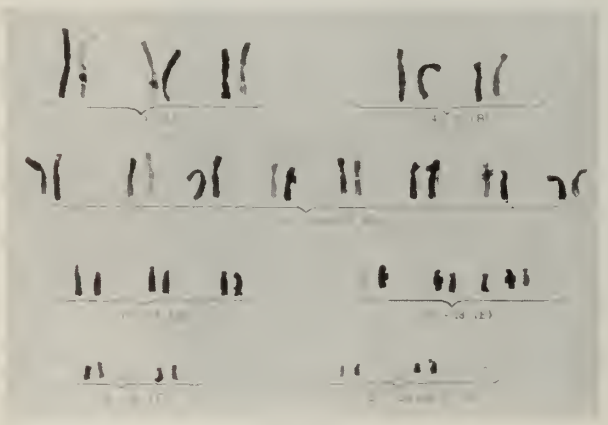


Fig. 4. Case I. Karyotype. Twenty-three pairs of chromosomes are shown. The extra chromosome (above E) is grouped between pairs 16, 17 and 18 because of the difficulty in determining to which pair the extra chromosome belongs.

Radiographic examination demonstrated small mandible and low-set external ears. (Fig. 5). The heart was enlarged with plethoric lung fields. The hands were held in flexion with marked ulnar deviation and overlap of the third and fourth fingers over the second and fifth (Fig. 6). The pelvis was small and average of the two acetabular angles measured twenty eight degrees (normal is twenty degrees). (Fig. 7) An intravenous urogram was normal. Chromosomal studies of peripheral blood leucocytes by a modification of the Moorehead method indicated 47 chromosomes with the extra one resembling the 17 and 18 pairs.

Clinical Course: The patient died at age 14 weeks with autopsy findings of a high

interventricular septal defect, a small patent foramen ovale, and deformity of bicuspid pulmonic and aortic valves. There was hypertrophy of both right and left ventricles.



Fig. 5. Case II. Typical facial appearance in trisomy 17/18. Note the low hair line, malformed low-set ears and hypoplasia of the mandible. Lateral roentgenographs demonstrate an elongated posterior fossa and macroglossia.

The lungs were congested. Gross and microscopic examination of the liver, spleen, pancreas, kidneys, brain, intestines, and uterus were all within normal limits.



Fig. 6. Case II. Ulna deviation of the entire hand with flexion deformity of the second finger at the proximal interphalangeal joint. The middle phalanx of the fifth finger and first metacarpal are short.

Comment

Clinically, the main abnormalities in the 17/18 syndrome are low-set and malformed ears, small face with micrognathia, hypoplasia of the orbital ridges and narrow palpebral fissures. The mouth is usually small and triangular, frequently with a high-arched palate. The thoracic and abdominal abnormalities are a short manubrium with a decreased number of sternal ossification centers at birth, partial agenesis or laterally tapered or tripartite clavicles, and hypermobile shoulders. The absence of discernable sternal ossification centers at birth has not been previously described. The ribs are slender, tapered and irregular. There is increased AP diameter of the chest. The heart is large, and most of these children have a ventricular septal defect and/or patent ductus arteriosus.

Other reported but less common abdominal findings are paraumbilical and inguinal

hernia, abnormal external genitalia, horseshoe kidneys, eventration of the diaphragm and imperforate anus.[5,6] The hands show ulnar deviation, are usually hypertonic and held flexed at the metacarpophalangeal and



Fig. 7. Case II. Forward rotation of the ilia with narrow iliac crest and large acetabular angles.

proximal interphalangeal joints but are normal distal to this point. Often there is a V-shaped gap between the forefinger and middle finger. The second finger tends to overlie the middle finger and the fifth finger overlies the ring-finger. Frequently, there is hypoplasia of the thumb and of all the nails.

There may be forward rotation of the ilia with narrow iliac crest and large acetabular angles in contradistinction to mongolism. There may be minor developmental anomalie such as curvature of the fibula.

The feet have a short first toe with frequent syndactyly of the second and third toes, often with a V-shaped gap between the toes, usually between the first and second toe. The proximal phalanx of the first toe is usually short or broad. There are often protuberant heels and varus deformities of the forefeet.

Summary

Trisomy 17/18 is a multisystemic syndrome of congenital anomalies which usually causes death in the first year. The radiologic manifestations may be more diagnostic than the somatic picture. The most common features are seen in the skull, chest, hands and feet. These are manifested by small mandible and mouth, hypoplasia at the orbital ridges, slender, tapered clavicles and ribs with poorly developed or non-ossified sternal centers. The heart is enlarged with plethoric lung fields suggesting a left-to-right shunt, most commonly a ventricular septal defect. There are short first toes, varus deformity of the feet, and overlap of the fingers in an ulnar deviated flexed position with V-shaped gap between the second and third digits.

Acknowledgment

The author wishes to thank Dr. Theodore E. Keats, Professor of Radiology, for criticism of the manuscript, and Dr. Robert F. Seldon, Jr., Assistant Professor of Pediatrics, University of Virginia Medical Center, for the photographs and chromosomal analysis in the case reports.

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Electrical Burns of the Mouth

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Electrical burns of the mouth occur usually in young children who bite into an extension cord or suck its exposed end. The characteristics of these burns are discussed and the treatment, both early and late, is presented. The prognosis is excellent.

ELECTRICAL BURNS of the mouth are not uncommon. They are seen most frequently in young children who, crawling unattended, bite into an electric cord or suck on the exposed end of an extension cord. Electrical burns in areas other than the mouth are also not infrequent, and are noted particularly in workers who accidentally expose themselves to high voltage wires or, especially in Europe, in children who grab unprotected heating bars with their hands. Electrical burns of the genitalia are also a problem in countries where uninsulated wires carrying high voltages pass near bridges or other high points. Occasionally, children will urinate on such wires, and the electricity will carry up the stream of urine to create a severe burn to the genitalia.

Electrical burns are of two types. The first is the arc type in which an arc is produced from the electrical contact to the body or between two electrodes external but adjacent to the body. This arc reaches a temperature of 2500-3000 degrees C. The second is the contact type of burn in which



Fig. 1-A. Child 2 weeks after burning mouth on extension cord, October, 1966.
B. Same child July, 1967 showing marked improvement with non-surgical treatment. Surgery can now be utilized for further cosmetic and functional improvement.

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the burn is caused by the current flowing through the body with some associated arcing at points of contact. In the contact type of electrical burn, the heat is generated by the resistance of the tissues to a flow of current (Joule effect).[1] An increase in the amount of current is accompanied by an increase in heat and tissue destruction. If the body's resistance were constant, the burn caused by either direct or indirect electrical current would be proportionate to the voltage. Deaths due to cardiac arrest are caused most easily by alternating current and may result from as little as 46 volts potential.

With short exposure, the possibility of death from ventricular fibrillation is greater with an increase of amperage up to a certain point, after which further increase in amperage results in a decreasing incidence.[2] At relatively high amperage, the likelihood of producing fibrillation is almost negligible.

Burns of the mouth present particular problems, inasmuch as they are exposed and in a moist area. Most burns in children are usually due to low voltage alternating current and are chiefly of the contact type with the electrical current passing through the tissues of the mouth either directly from the metal electrodes of an electrical plug or through the tissues and the saliva which bridges the gap between the electrodes and the mouth. The saliva-tissue combination offers less resistance than either medium by itself.



Fig. 2. A. & B. 13 year old girl with mouth scars from burns during infancy.
C. Same patient after a single surgical procedure.
NOTE: Angles of mouth are symmetrical.

These burns have often been attributed to the child chewing on an electric cord lying on the floor. Thompson[1] and Fleury[5] point out that the vast majority of these burns are due to the child sucking on the end of an extension cord, the other end of which is plugged into a wall outlet. In Thompson's series 37 of 43 children received their burns in this fashion while all five infants in Fleury's series were burned while sucking on the end of an extension cord. This has also been true in the majority of our cases (17 out of 21) but at least one infant was burned while chewing on the central portion of a cord without any ap-

parent damage to the cord noted after the child's injury. If it is universally true that most of the burns are received when the child places the end of the extension cord in his mouth, most of the electrical burns of the mouth in children could theoretically be prevented by either not using loose extension cords in an area occupied by children under the age of five or by fastening the plug to the wall or other object so that the child would be unable to get it into his mouth. Occasionally, the current will pass through the child's mouth to another electrical contact, i.e., a radiator or pipe serving as a ground. In these cases, electrocution may result but in 95% of electrical injuries to children's mouths the oral burn is the only complication. The electricity will be transmitted in various ways by the different densities of the tissue and the moistness of the mouth. Extensive damage can develop under intact skin and occasionally muscle and nerve necrosis cannot be diagnosed early because the skin surface appears uninvolved.[3]

The typical early burn is usually noted to have a central area of lip tissue destruction, surrounded by a chalky white area of insensitive skin, with a circular red weeping area around the periphery. Actual damage and destruction of tooth buds, mucous membrane, skin, muscles, nerves and bone may occur. After initial burning, the wound does not appear to be sensitive and, indeed, children feed surprisingly well with half of the lip ulcerated and destroyed. The appearance of the electrical burn wound is deceiving in two ways. First, the actual extent of initial damage cannot be ascertained for several days due to unknown subcutaneous tissue damage. Secondly, after accurate demarcation and sloughing are evident, usually in approximately two to three weeks, the degree of tissue loss frequently seems enormous and parents express the thought that the child has lost all of the lip. Because of the remarkable healing in the area, this type of oral wound rebuilds amazingly well, and very often a severe ulcerated deformity will end as only a small scar of the vermilion. For these reasons, we advocate conservative treatment of the initial burn.

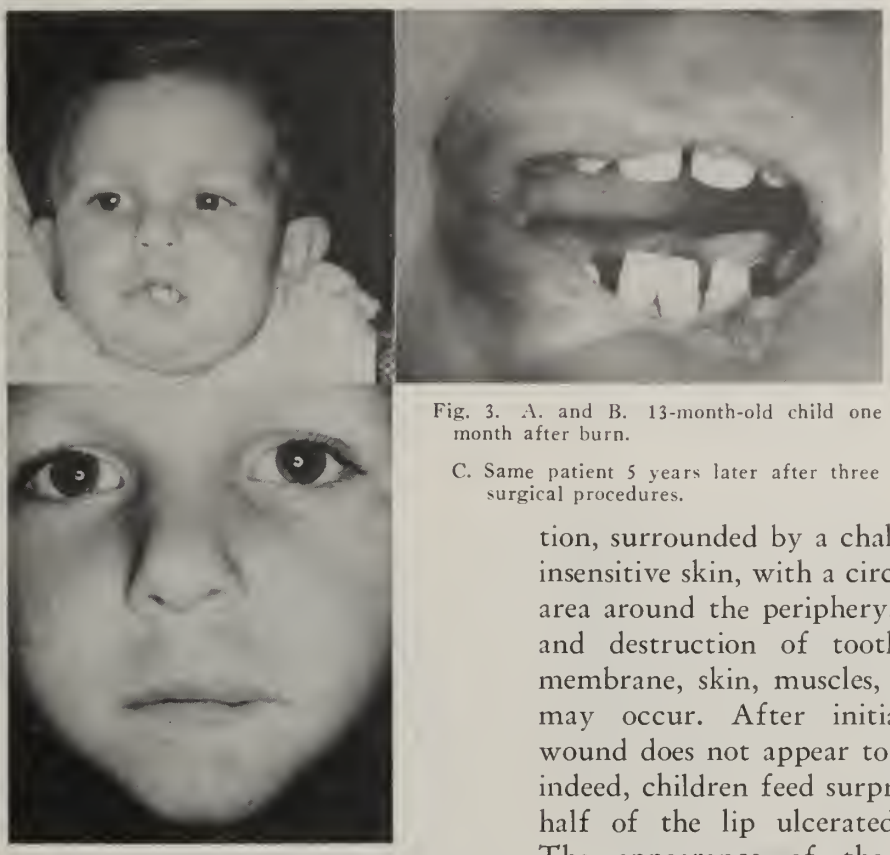


Fig. 3. A. and B. 13-month-old child one month after burn.

C. Same patient 5 years later after three surgical procedures.

Initial Treatment

When the burn is first diagnosed, the child should be checked systemically to

make certain no other points of electrical current entrance or exit are present.

The oral wound should be covered with an antibacterial ointment, and systemic antibiotics should be administered to prevent extension of the damage by wound infection. The mouth should be cleaned several times daily with peroxide or saline, and all obviously necrotic tissue should be removed. Only in extensive burns is it necessary to hospitalize the patient. Home care by the parents is adequate in most cases.

The burn will usually demarcate, and the necrotic tissue slough and be discarded by the second or third week. Red granulation tissue will then appear, and the lip will heal slowly with scarring. After the initial necrotic tissue is removed, systemic antibiotics are not necessary. Continued local care of the wound is advised. If bone sequestrates, or if a tooth loosens, the dead material should be removed. The wound will heal in four to six weeks and re-epithelialize completely.



Fig. 4. A. shows narrowing of the mouth following an electrical burn.
B. The same patient one year later after surgery.

Secondary Treatment

When healing has ceased in two to three months following the burn, the lip and mouth problem should be re-assessed. Ordinarily, the tissue deficit will then appear half to three-quarters less than the original burn. Scar contractures will invariably distort the mouth and shorten the commissure of the lips, however. Frequently, the sulcus of the mouth will be destroyed, and saliva drooling will be a constant irritant. The lip width may be lessened to such a degree that eating may be impaired. At this time, it is difficult to resist the plea of the parents for immediate reconstruction. Only a few surgeons advocate early repair of electrical burns of the mouth.[5] The considered opinion of the overwhelming majority is to defer surgery until the scars soften to make definitive reconstruction feasible and to obtain a better result.[4-6] Usually, we recommend reconstruction at six months or more post injury. A minimum of six months post burn is required for adequate softening of the damaged area.

Reconstruction

The patient should be admitted to the hospital for general anesthesia and surgery. The angle of the mouth should be opened, scar tissue excised, and the width and commissure of the lips made normal. External scars should be excised and closed with flaps or Z-plasties as indicated. Mucous membrane can be obtained from inside the

mouth, and local flaps elevated and transposed to reconstruct the vermilion. A lateral flap should be interposed at the commissure to interrupt a circular repair scar line to prevent contraction. Mucous membrane grafts to restore the upper or lower sulcus of the mouth may be required in a subsequent procedure. Frequently, it is not possible to perform a complete reconstruction in one stage, and the parents should be advised of the difficulties inherent in this type of repair. In most instances, parents are cooperative and appreciate any attempt to revise the serious deformities.

If bone substance is lost, and if teeth have been damaged irreparably, reconstruction by the use of a prosthetic appliance may be indicated as the child grows older.

Prognosis

While a perfectly normal appearance of the mouth cannot be achieved in all cases, the over-all results of reparative surgery are excellent in this condition. It does not matter at what age the scar is seen for repair, and if previous attempts are less than ideal, repeated surgery can be performed as the patient grows older and larger. With adequate and competent repair, the stigmata of a small, tight, scarred and deformed mouth can be avoided.

Summary and Conclusions

1. Electrical burns of the mouth are described.
2. The etiology of electrical burns of the mouth is discussed.
3. A conservative initial treatment is outlined for early cases.
4. Secondary reconstruction of burn scars of the mouth is advocated.
5. The prognosis is excellent for full functional and cosmetic recovery.

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Let's Reminisce!

THE FAMILY PHYSICIAN—We take it to be the duty of the family physician, not only to answer the calls of his patient, and then and there give his advice, but also to suggest to parents the manner of treatment and method for the conduct of their children. We would say to a young couple to employ an active, intelligent, well-read and practical physician. Be certain that he is a gentleman. Let the physician grow up with the family. Let the children look upon the physician as next to priest or pastor of the family. He should be consulted frequently, not only when disease forces, but when there is no indication of disease. In the matter of schooling, athletic exercises, amusements and *work* of the child let the physician be consulted. The parent does half his duty when he calls in a doctor to prescribe for diarrhea and scarlet fever. Even if there is health or apparent health, the doctor should be called in and his advice followed. (Virginia Medical Monthly, February, 1882)

Clinicopathological Conference . . . .

Liver Failure in a Young Man

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CLINICAL DISCUSSANT:

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Assistant Professor of Internal Medicine

L. N. A. UVH # 58-48-03
Autopsy No. 11308

This conference was held December 2, 1967.

Clinical History

This 18-year-old, unmarried furniture sander was referred to the University Hospital on September 5, 1967, for evaluation of ascites and edema of at least two months' duration. Seven months previously he had consulted a physician for sore throat; splenomegaly was noted, and a heterophile test for mononucleosis was negative. He was not seen again until he developed dark urine, mild diarrhea, abdominal swelling, and dependent edema. Treatment with tetracycline was ineffectual, and he was referred to a local hospital for more definitive diagnosis. At that hospital he was found to have slight cervical adenopathy, enlargement of the liver and spleen, ascites, and pretibial edema. Laboratory studies there revealed hypoalbuminuria without proteinuria, elevation of the SGOTransaminase to 75 units, prolongation of prothrombin time to 27 seconds with a 12-second control, and elevation of alkaline phosphatase to 8.7 Bodansky units. Serum bilirubin was normal. Liver biopsy was attempted twice without success. Treatment with furosemide resulted in diuresis, with decrease in ascites. Two weeks prior to admission here there was an episode of fever, cough and sore throat, which responded to treatment with tetracycline. One week before admission he developed cramping, lower abdominal pain.

There had been no previous history of definite jaundice or light stools. He had occasionally used chemical solvents, but there had been no exposure to carbon tetrachloride, or animal or insect bite. There had been no rash or arthritis. There was no family history of liver disease. He had never been ataxic or shown obvious neurologic abnormalities.

Physical Examination here showed a dark-complexioned, chronically ill-appearing young man without fever. Blood pressure 125/80, pulse 80 and regular. There was cloudiness of the corneal margins, and this raised the question of whether a Kayser-Fleischer ring was present. This question was never definitely answered. There was mild jaundice. There were dullness and diminished breath sounds at the right lung base, and scattered rales above this were present. The heart was not enlarged; an ejection systolic murmur was heard along the left sternal border without other abnormal cardiac findings. Ascites, marked hepatomegaly and splenomegaly were present. Neurological examination was normal, except for slight tremor of the outstretched hands. No cause for the abdominal discomfort was found.

Laboratory findings on admission were Hct 35%, WBC 8600 with a normal smear, reticulocyte count 2.7%, and platelets 98,000/mm^3. The urine contained a trace of bile and the stool was positive for occult blood. Total serum bilirubin was initially 3.95 mg.% (direct 0.49 mg%), prothrombin time 13.5/11.5 seconds, alkaline phosphatase 3.6 units, lactic dehydrogenase 110 (normal up to 100), SGOTransaminase 178 (normal up to 50 units), SGPT 0. Serum

albumin 2.5, globulin 2.1 Gm.%, cholesterol 85 mg%, uric acid 5.0 mg%, and bromsulphalein retention 48% at 45 minutes. Blood sugar was 76 mg%, urea 40 mg%. Serum calcium 8.0 mg%, phosphate 5.4 mg%. Partial thromboplastin time was prolonged. Serum electrolytes were normal except for a serum potassium of 2.9 mEq/L.

Hospital course: On the second hospital day the patient became lethargic and developed a flapping tremor of the outstretched hand. Treatment consisted of protein restriction, oral neomycin and potassium supplements, and cathartics. The hypokalemia was corrected, but the encephalopathy worsened. Prednisone, 40 mg. per day, was added. The hematocrit fell to 20% without evidence of significant gastrointestinal bleeding, and platelets fell to 6000/mm^3. Rapidly increasing jaundice and diminishing urine output were followed by death in coma on the fifth hospital day.

Clinical Discussion

Dr. David Stone: As those of you who have read the protocol today have recognized, this young man presented us with major problems in diagnosis and treatment. Another facet of this case is that our terminology and diagnostic criteria may also have to come under some scrutiny, since we may have as much of a problem in labeling his disease as we do in predicting the pathology. The differentiation between various types of parenchymal liver disease is of some therapeutic importance now, whereas in the past this was not the case. Formerly we had no real way of approaching a patient with liver disease except by the broadest of supportive techniques. Currently, however, there are at least one or two varieties of this problem that we can approach more or less specifically. Our major problem in parenchymal liver disease is not recognizing the pathology, which is often fairly predictable, but in understanding etiology and pathogenesis. Until we can arrive at etiology and pathogenesis through our clinical acumen we can be of only limited benefit to patients like this one.

It will be unnecessary to go through the details of the protocol. As noted, they demonstrate rapidly progressive liver failure, beginning at an unknown time but certainly exacerbating in July of 1967. We will discuss later the events leading into his terminal episode.

The findings on physical examination again are not very significant as far as etiologic concepts are concerned, with the exception of the possible Kayser-Fleischer ring, which will have considerable importance, and to which we will also return. He did have a right pleural effusion and perhaps pneumonia on the right, and what I assume to be a functional murmur, but these apparently do not have immediate importance to our problem. It should be noted, I think, that his neurologic examination was completely normal, and this will have very definite importance at a later point.

His laboratory findings are interesting. His initial hematocrit was 35% and his reticulocyte count was 2.7%, indicating some reason for anemia and marrow hyperactivity, though we cannot be sure whether there was blood loss or hemolysis at this stage. We are told that his urine had a trace of bile. The prothrombin time was amazingly normal on admission here compared to what it had been outside. It might be well to point out that the prothrombin time of the magnitude found prior to admission should be a clear-cut warning of the marked severity of the disease this boy had. This degree of elevation is seen only with severe liver cell impairment, assuming that Vitamin K supplies are adequate. There are a few other findings which are interesting, notably the normal uric acid level, the slightly low serum calcium, and the mildly elevated phosphorus. I cannot tell you why his SGOT was so elevated at 178 and his SGPT 0. At this stage we know that his clotting factors were abnormal by virtue of the abnormal partial thromboplastin

time. His last days were complicated by a fall in hematocrit to 20% at a time when his stool showed only a 1+ reaction to guaiac, and one has to wonder again if he might not have had a hemolytic episode. We might see the x-rays here to see if they can help us.

Dr. Torbjorn Westgaard: These films will tell you more about the effects of this man's liver disease than they will about its cause. The chest x-rays show large amounts of pleural fluid bilaterally, extending into the inter-lobar fissure on the right. The cardiac silhouette is obscured by elevation of the diaphragms and the pleural fluid. One could not exclude pericardial restriction or other causes of such extensive fluid accumulation, but advanced liver disease may certainly produce a picture such as this. The abdominal films show a homogeneous density throughout and bulging flanks, characteristics of ascites. Enlargement of the liver and spleen are present.

Dr. David Stone: The two major causes of severe primary hepatic failure are cirrhosis and hepatitis, and I think it would be well to discuss what we mean when we use these two labels. They certainly do not reflect specific processes, the first indicating hepatic inflammatory disease with necrosis and falling out of liver cells, and the second dealing with scarring and fibrosis of the liver associated with parenchymal regeneration and hepatocyte dysfunction. The first thing anyone looking at this protocol would consider would be the possibility that this boy did have some type of hepatitis, and that he had gone into a chronic, relentless stage. One might wonder about there having been an episode of anicteric or unrecognized icteric hepatitis sometime in the past. We certainly get the impression that his liver disease began before March of 1967, because at that time his spleen was enlarged. We can suspect that whatever process was going on had been present for a significant period preceding this. We have all heard allusion made to chronic, presumably viral hepatitis, which is jaundice and evidence of hepatitis existing beyond three months, and certainly our protocol would fit fairly well with this. We are told that he has had occasion to be exposed to chemicals, and this is always intriguing as far as possible toxic factors are concerned. We know that there are a variety of agents which can cause liver cell death, notably those compounds akin to iproniazid and those solvents similar to carbon tetrachloride. We know that on occasion these processes associated with cell necrosis may progress to hepatic scarring and cirrhosis.

Another group of considerations might be lumped together under the term, "juvenile cirrhosis". Perhaps this is not a separate group from those which might fall into the post-hepatitic or post-necrotic categories, or even separate from those cases which have been called chronic hepatitis, plasma cell hepatitis, "lupoid hepatitis', etc. These are clinical states in which some attempt has been made at correlation with pathogenetic activities.[1] This group of cirrhoses usually has a subacute onset, often with no clear-cut etiology or preceding illness. Clinically, individuals with these diseases may look quite well for long periods of time. They may have associated findings which we have become accustomed to seeing in diseases of autoimmunity, notably arthritis, skin rashes, and even ulcerative colitis. They may show evidence of alterations in immune globulin response with greatly elevated gamma globulin levels and positive LE preps. From this it should be apparent that our patient today may have a rather limited potential as far as pathologic findings, but the etiologic possibilities in his disease may be diverse.

The last problem I would like to discuss in relation to etiology is Wilson's disease. I think it is fair to say that if a Kayser-Fleischer ring were indeed present, then the patient had Wilson's disease without equivocation. This patient apparently did not have the athetoid motions and other characteristic neurologic changes. Neverthe-

less, the studies of Kehrer,[2] Andre,[3] and Chalmers,[4] have shown that liver disease may precede the neurologic abnormalities by long periods of time, and indeed may be the only manifestation of the disease process. The liver disease of hepatolenticular degeneration may be no different clinically or pathologically from so-called juvenile cirrhosis or active chronic hepatitis, pointing up again that once the common pathway or irreversible parenchymal cell disease has been reached, etiology may no longer be discernible. Wilson's disease is inherited as an autosomal recessive. We have to assume that our patient was a homozygote with siblings or other members of his family who were heterozygotes, or that he was a mutation, since there is no family history of neurologic or liver disease. In Wilson's disease serum levels of caeruloplasmin are markedly decreased in over 90% of patients studied. Interestingly there have been fourteen patients reported with normal caeruloplasmin levels at one time or another.[5] In addition, the decreased caeruloplasmin levels may not be pathognomic since about 20% of heterozygotes may also have low values.[6] There is an increase in copper in various tissues of the body, notably the liver, the brain, and the cornea. There is usually an increase in hepatic copper content to greater than 250 micrograms per gram of dry liver. Renal tubular defects have been described with excess loss of uric acid, frequent hypouricemia, hypercalciuria, and quite commonly aminoaciduria. Interestingly, as far as our patient is concerned, there has been a rare presentation with hemolytic crisis.

As I noted, up until fifteen years or so ago, the question of the type of liver disease was largely academic, most of these progressing inexorably no matter what therapy was offered. Presently it is of some practical importance to delineate the pathogenesis of each problem we see, because at least two of these syndromes may be amenable to palliation, if not definitive treatment. It has been shown, for example, that in patients with Wilson's disease the neurologic deficits may sometimes be dramatically corrected by the use of copper-binding agents, such as BAL or penicillamine. If it were possible to be more successful in case finding, it is conceivable that early treatment with such agents might also prevent or reduce initial toxic effects on liver cells, and do a great deal to prevent one of the lethal outcomes of Wilson's disease. In another situation it is fairly well established now that steroid therapy in those patients with so-called juvenile cirrhosis may be of value in improving their functional capacity.[1] There is no evidence that steroid therapy in these patients prolongs life, but certainly the symptoms may be reduced, and jaundice and enzyme elevations may significantly decrease. Thus, an increasing awareness of etiologic diversity may make it possible to offer patients with parenchymal liver disease more definitive therapy before the final common pathway of irreversible cirrhosis is reached. Perhaps an early measurement of this man's ceruloplasmin level and successful biopsy would have led to a trial of some specific form of treatment.

I doubt that I can make a definitive statement regarding this boy's terminal event. I am intrigued with the possibility that he may have had a hemolytic process, perhaps causing his abdominal pain, and perhaps being exacerbated during his last two days. There is certainly not very good support for this, except for the falling hematocrit and reticulocytosis. We know that individuals with parenchymal liver disease are quite susceptible to infection and are extraordinarily incapable of handling infectious processes well. It could easily be that a low-grade pneumonia present above the pleural effusion on the right is what tipped this boy into his coma and death. His renal failure surely contributed to his rapid demise. I do not think it is valid to speak of the "hepatorenal syndrome" as a pathologic entity, since there have never been any definite renal parenchymal changes directly

attributable to liver disease. However, there certainly is a very real clinical association between the failing liver and the failing kidney. Formerly it was thought that renal functional changes in hepatic failure reflected a decrease in plasma volume with a consequent diminution in glomerular filtration rate and renal plasma flow. More recent studies published in the last several months have indicated fairly clearly that there is no consistent decrease in any of these parameters, and therapy utilizing volume expansion has been only minimally helpful in reversing or altering the oliguria.[7]

In summary, then, I would have to say that this young man had "juvenile" cirrhosis without being able to elaborate on its etiology. I cannot make a diagnosis of Wilson's disease, given the information in the protocol, though certainly a Kayser-Fleischer ring would make that diagnosis. I suspect that Dr. Smith will show us a multinodular or post-necrotic cirrhosis, perhaps with some evidence of subacute inflammation. He may also show a small area of pneumonia above the pleural effusion on the right and there will likely be hydropic degeneration and bile-plugging of the renal tubules.

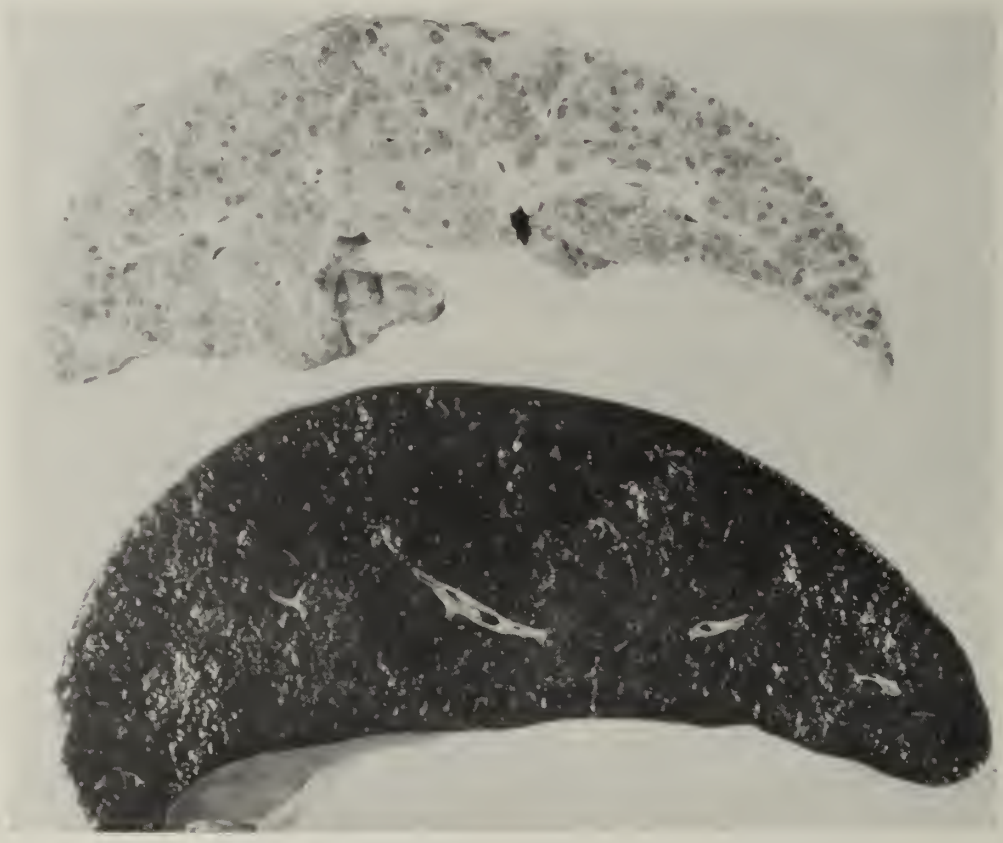


Fig. 1. Longitudinal sections of the liver and spleen. The coarsely nodular, cirrhotic liver was smaller than the huge congested spleen.

Dr. Stone's Diagnosis:

1. *"Juvenile cirrhosis", possibly due to atypical Wilson's disease.*

Pathological Discussion

Dr. David E. Smith: There were an ascites of 2,000 ml. and a rather remarkable situation of the liver and spleen at autopsy (Fig. 1): the liver was reduced in size to 1,000 grams, while the spleen was enlarged to 1,400 grams, a reversal of the usual weights. The liver had a coarsely nodular configuration and on the cut section showed distinct, broad, fibrous bands surrounding regenerative nodules. The nodules were large,

up to 1 cm. or so in diameter, and were of variegated colors, some being obviously necrotic. The gross appearance of the liver was that of multilobular cirrhosis, a term synonymous with what Dr. Edward Gall calls post-hepatitic cirrhosis, and used interchangeably by many with the term "post-necrotic cirrhosis", although I prefer to reserve the latter term for the cases in which there are large areas, several centimeters or so in breadth, of total hepatic destruction. As Dr. Stone has pointed out, the classification of a cirrhosis on an anatomic basis, regardless of what terms are used, is all too often of little specificity or help in elucidating the still-unknown mechanisms of the etiology and pathogenesis of many cases of cirrhosis. Certainly, the appearances of this liver place it in such a nonspecific category.

Microscopically (Fig. 2) there was a mixture of features typical of various types of cirrhosis. There was a little fat in some of the hepatic cells, and a few branches of portal spaces or central veins could be found in the nodules surrounded by the larger fibrous scars. The hepatic cells themselves contained none of the cytoplasmic coagulative changes of the types that are to be found in either a nutritional cirrhosis or active viral hepatitis. Many of the lobules were almost totally necrotic. This was undoubtedly a feature of the terminal vascular collapse. Exactly what triggers this appearance of necrosis of lobules in terminal cirrhosis is not always easy to determine, and there are few clues in the present protocol except for the decline of the hematocrit to 20 per cent.

The intestine was covered by a greyish fibrosis of the serosa, a reaction to the chronic ascites and an additional evidence that this man's hepatic disease extended considerably beyond the date first recited in the protocol. The kidneys were enlarged and icteric. On microscopic section there were most dramatic crystals and casts in the proximal convoluted tubules (Fig. 3). Some of these crystals were thin, tubular or cylindrical and brown, others were round, doubly refractile, and of the type generally recognized as leucine crystals. In other tubules there were bile-stained casts, and casts which stained positively for iron. I am suspicious that the thin, cylindrical crystals are amino-acids, as were the more obvious round crystals of leucine. These were con-

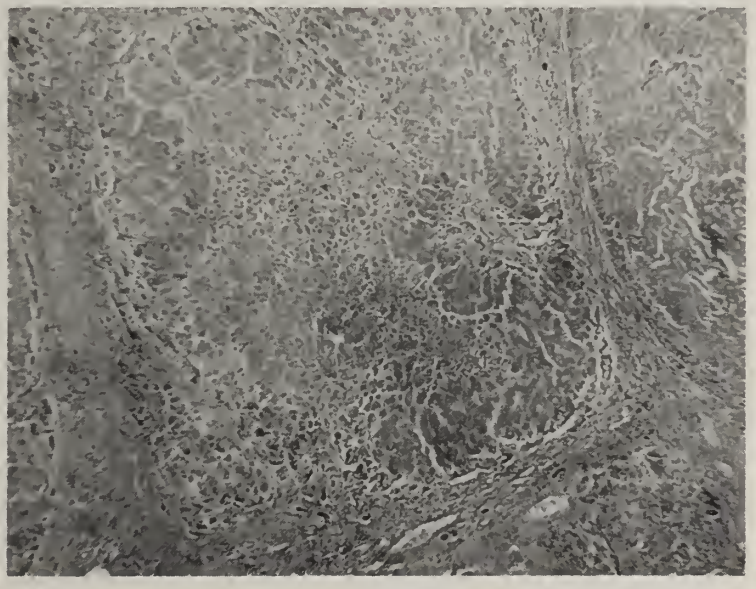


Fig. 2. An irregular regenerated nodule in the multilobular or "post-necrotic" cirrhosis. There are zones of acute necrosis as well as the chronic features of broad fibrous septa and proliferated cholangioles. (Hematoxylin and eosin stain; X50.)

centrated in the renal tubules because of the foci of acute necrosis in the liver, but they may also have indicated a more chronic amino-aciduria that would fit nicely into our concept of some of the hepatic diseases discussed by Dr. Stone. At any rate, the various crystals and numerous casts were accompanied by cellular changes in the tubules that were sufficient to correlate with the terminal evidences of renal insufficiency in this patient.

The brain was normal to inspection externally and on multiple cross sections. Microscopically, however, there were typical Alzheimer Type II cells in many locations (Fig. 4), particularly in the globus pallidus, the putamen, and the caudate nuclei. These enlarged glial nuclei were of a typical rather

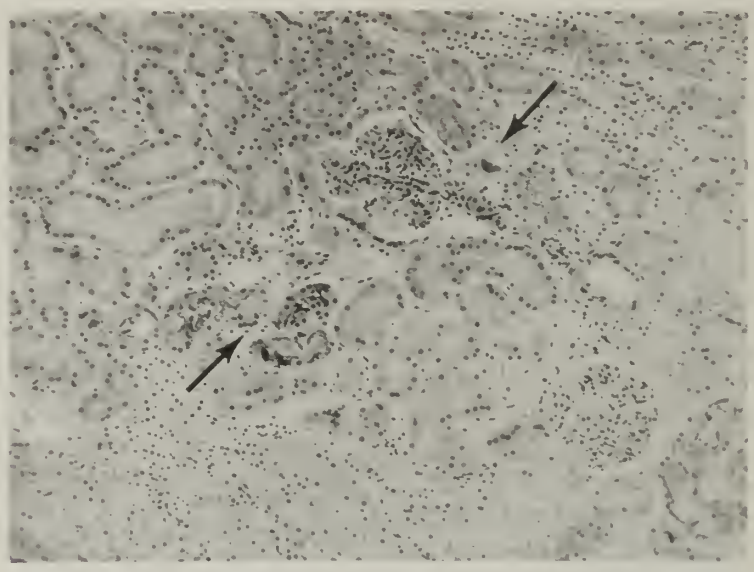


Fig. 3. Kidney with round leucine crystals (upper arrow) and tubular brown crystals (lower arrow) in proximal convoluted tubules. These indicate an amino-aciduria partially the result of necrosis in the liver, but they may also have been related to a characteristic metabolic defect of Wilson's disease. Elsewhere there were typical casts and lesions of acute tubular necrosis. (Hematoxylin and eosin stain; X 130.)

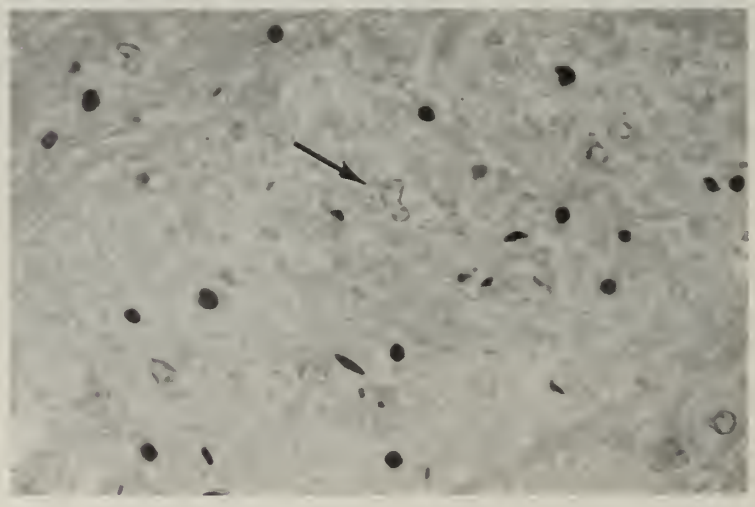


Fig. 4. Type I Alzheimer glial nuclei in the caudate nucleus. The typical folded contours and delicate chromatin characterize cells that may occur in various other types of hepatic disease as well as Wilson's disease. (Hematoxylin and eosin stain; X 600.)

delicate appearance and irregular contour. We have learned to recognize that they are not specific for Wilson's disease but may be found in many types of hepatic failure. The Alzheimer Type I glial cell and Opalski cell, which are considered more specific for Wilson's disease, were not present in this material. An additional finding of considerable interest was a very pale central body surrounded by a clear zone within the nucleus that had the general appearance of an inclusion body. These were never very dense or clearly outlined as is characteristic of viral inclusions. Although the immediate temptation is to suspect that they might be the effects of a virus that is also working on the liver, it should also be remembered that heavy metals, such as lead and antimony, are capable of causing the formation of intranuclear inclusions in cells of the liver and kidney. As such, we would correlate these bodies with the mineral disturbances, which seem to be a central portion of the syndrome of Wilson's disease.

A section of the cornea of the eye at the limbus contained no evidence of pigment granules in Decemet's membrane. There was also a small paravertebral abscess that was present behind the pleura on the right side in the midthoracic region. It was of interest simply in proving the existence in this patient of an acute infection. There was very little pneumonia or other evidence of infectious processes, but as Dr. Stone has pointed out, a minimum of infection can be important in the decline of patients with advanced hepatic disease.

Tissues from this case were submitted to analysis by atomic absorption spectometry by Dr. Martin G. Netsky, who is conducting a survey and research program utilizing this technic with Dr. Willard Harrison of the Department of Chemistry. Their methods allow the extremely sensitive detection of very small amounts of the trace elements. They have been establishing base lines in various tissues, particularly parts of the central nervous system, and this case is one of the most dramatic deviations from normals that they have encountered. They demonstrated marked increases in copper (three to five fold above normal) in various structures of the brain, particularly in the grey matter of the basal nuclei and in the liver. There were also increased amounts of zinc in the basal nuclei and decreased amounts of iron in both the brain and liver.

The abnormally high concentrations of copper in the brain and liver are central to our present concepts of the nature of this case. Wilson's disease, after passing through stages of gross pathological definition, clinical definition, and a chemical definition based upon the presence of low levels of ceruloplasmin, has now arrived at a point at which the central feature is thought to be a disturbance of the amount of copper in the tissues. This case certainly fits into that definition and has, of course, other supporting features: the atypical cirrhosis, the microscopic changes in the brain, and the lack of a clinical history of either hepatitis or nutritional disturbance. It now appears that this disease is basically a disorder in which copper is abnormally concentrated in the brain and liver and is associated, probably by acting as an endogenous toxic agent, with structural changes in those organs. This may well not be our final definition of Wilson's disease, for our concepts have continuously outgrown criteria previously offered for its definition. I should point out that this particular case lacks a number of features that we would like to have included at one time or another in the definition of Wilson's disease. There was no familial history, there were no gross pathologic changes in the brain, there was no history of a chronic, clinical neurological disorder, there was no Kaiser-Fleischer ring, and the ceruloplasmin levels, which were reported after death, were within normal range. Nevertheless, we are left with the fundamental features of high copper levels in the tissue and compatible structural changes in the brain and liver. Our final categorization

of this case has consequently been "atypical Wilson's disease".

Anatomical Diagnosis:

Multilobular cirrhosis with highly elevated copper content in the liver and brain.
Alzheimer's glia in the basal nuclei.
Splenomegaly, ascites, and icterus.
Tubular necrosis with amino-acid crystals in the tubules of the kidney.
Small right paravertebral abscess.

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Correspondence . . . .

Medicine's Concealed Hemorrhage.

To The Editor:

The purpose of this letter is to briefly express the opinion that the Guest Editorial appearing in the February issue of the Virginia Medical Monthly entitled "Medicine's Concealed Hemorrhage" was in extremely bad taste.

I believe the Medical Society of Virginia has done a great disservice to itself and the hospital administrators of this Commonwealth in printing this garbage. We are disappointed that your editorial policy apparently endorses this type of journalism.

Sincerely,

Stuart D. Ogren
Executive Director
Virginia Hospital Association

To the Editor:

In reference to your Guest Editorial in the February issue of the *Virginia Medical Monthly;* all I have to say is, "for shame, for shame."

To allow such garbage to be printed in such a fine magazine is a discredit to the Virginia Medical Society.

Yours very truly,

William H. Flannagan
Director
Roanoke Memorial Hospitals

February 26, 1968

Mental Health . . . .

Vocational Rehabilitation Unit Central State Hospital, Petersburg Eighteen Months' Experience

A. RAY DAWSON, M.D.

The rehabilitation concept is not new. Hannibal had his restoration camps; both the Germans and the French developed rehabilitation activities during the Franco-Prussian War; England had one of its largest restoration camps in Egypt early in World War I.

During World War I and after, military medicine in this country included rehabilitation. Rehabilitation started its rapid growth during World War II and was made necessary by other developments in medicine, primarily the controlling of infections, but also as a result of newer procedures in surgery. Now, medicine has the capacity to save many lives, but often this results at the cost of a residual disability.

When one reviews the history of the rehabilitation concept in the field of mental and emotional disturbances, certain basic facts in the history come in view. Occupational therapy and work therapy are not new in the treatment of mental disorders. Dr. Winick[1] traces work therapy as a treatment for mental ills as far back as Hippocrates; it perhaps goes back even further than this. However, the present rehabilitation concept did not take firm root until drug therapy and other newer methods of treatment created the situation where rehabilitation now becomes imperative.

This report is based on 18 months' operation of the newly-established rehabilitation unit at Central State Hospital, Petersburg, but in fairness to our consideration, I estimate that efficient operation should be in terms of 10 to 12 months.

Dawson, A. Ray, M.D., *Director of Rehabilitation, Department of Mental Hygiene and Hospitals.*

Approved for publication by the Commissioner, Department of Mental Hygiene and Hospitals.

First, the Department of Vocational Rehabilitation experienced much difficulty in employing persons with required skills to fill the necessary positions, as these people are in scarce supply. Second, the hospital went through the transition from partial racial integration to full integration during this period, and although this transition was without major incident—in fact, very smooth—it certainly was not without prior anxious planning and most careful time-consuming execution. This report will deal with our actual experiences and some conclusions which seemed to us to be valid.

As a result of a joint agreement between the Department of Mental Hygiene and Hospitals and the Department of Vocational Rehabilitation, a building housing approximately 100 patients with office space, dining and recreational facilities, plus evaluation shops, was set aside for rehabilitation patient-clients. This building was titled "CHAP (Community Home Adjustment Program) House". Patients selected for housing in this facility required evaluation, training and resocialization in much more depth than the average cases that the vocational counselor worked with on the ward and then followed into the community for job training and/or placement. This in-depth rehabilitation endeavor was what was added as mentioned in our article of September, 1966.[2] In addition, a vocational workshop was opened August 16, 1967. The two above facilities are the subject of this report.

The use of the housing facilities was delayed in development, with an average occupancy of about 30 patients until the spring of 1967. The census then started to grow, reaching 85 in October, 1967, where it has remained plus or minus a few. Ancillary facilities will be quite crowded with a patient load over 90.

The vocational workshop program was established primarily for the sicker ward patients with the hope that this experience would restore the patient's confidence in himself (build ego strength) and develop work habits, these to be used as stepping stones to evaluation and training in depth including transfer to CHAP House, if indicated. For the last few months, the CHAP shop has been serving between 40 and 50 patients a day. The effectiveness of the shop has exceeded expectations.

The reader is referred to an article by Dr. Hansen[3] that appeared in the February, 1968, issue of the Virginia Medical Monthly relative to the excellent shop and community work program which has been going on at Western State Hospital for some three years.

Patient selections for the rehabilitation facilities (housing and workshop) are based upon the ward physician's recommendations and selection by a joint hospital-vocational rehabilitation board. Certain criteria of both must be met. Two hundred and fifteen patients were admitted to CHAP House over the 18-month operational period, with 90 of these during the last six months. A few patients were returned to the ward for further treatment. Some of the patients furloughed could not be followed because of moving out of the state. A few patients had irregular discharges, and a few were discharged to their families without follow up. The above have been lost to our consideration. Fifty-six have been furloughed or discharged to employment and have been followed in employment. Sixty-four, following furlough, have been transferred to their local community vocational counselor for follow up; our reports indicate that these are now employed or receiving rehabilitation services. Ten of the furloughed patients have been rehospitalized at Central State Hospital, only two of whom have remained in the hospital for profound psychoses. This figure of 10 cannot be accepted as a true readmission rate because of the fact of time and also because some cases have been lost to our study. However, as these cases were followed up in work situations and community settings, we were encouraged to believe that the readmission rate would be reduced. Experience gained leads us to conclude that intimate post-hospital follow up is a most important part of the restorative process in its entirety.

As a part of work training, patients are put on jobs in the city of Petersburg. For the last few months, an average of 15 patients have been placed in full-time employment in town. These patients use the regular bus service, carry their lunches, and in all other respects, are regular employees returning to CHAP House at night. This is usually the last step before furlough. Our experience in work-placement parallels the experiences of others in that this procedure works very well.

Certain features of the psychotic and post-psychotic state, which have been written about in numerous articles in great detail, become quite evident in the rehabilitation setting, among which are the following:

(1) The ability to do productive work is the last ability to go in a psychosis and the first ability to return in recovery. I have seen patients actively hallucinating while doing productive, repetitive work and was told that the productive level and the accuracy level were above average;

(2) As you watch these patients grope for normality in a productive or social setting, one must conclude that an acute psychosis is a very frightening, unreal personal experience, and

(3) The road to recovery is a rock-strewn highway, being characterized by slow progress and often subjected to most painful recall.

Although all patients transferred to CHAP House must express a desire for same and have had full ground privileges for a sufficient time to determine stabilization, this change is a frightening experience to

many patients, some of whom, after a few days, request return to the protection of their old wards. In order to assist the patient in adjusting to CHAP House, which would seem to be a minor transition, a welcoming committee of patients has been established. Someone from this committee spends most of the first two days with the anxious patient and further guides and helps him for the first week or ten days. Even with this welcoming committee and staff support, some patients have felt too uncomfortable to remain.

Ego strength seems to return more rapidly in a work setting than a social setting. In fact, we have seen work proficiency increase while at the same time, social aptitude decreases. As would be expected, social restoration becomes a large and important part of living in CHAP House and will be discussed later. Freud[4] has stated that "the ego is first and foremost a body ego". If this is true, then one must assume that by slow growth or rapid onset, the shattering of the body concept and the concept of its surrounding environment is the basic process in a profound psychosis. It is most interesting during the recovery process to observe that as patients develop working and learning skills, they develop interest and growth in social skills. Perhaps work and classroom instruction are normal to us all, and if the body reacts normally in a normal setting, there must be a carryover in other areas.

The process of personality reintegration, keeping in mind the above, is the foundation on which the rehabilitation of the psychotic and post-psychotic is built. The evaluation of abilities and particularly potential abilities can never be hurried. Some have referred to the psychotic as being childlike and in many respects they are, with faith, hope, and desire for recognition and love prominent in their daily activities. If one watches these patients learn or relearn work and social skills, one is reminded of watching a child fill his UNIVAC (medically referred to as the cortex and adjacent cranial structures) with experiences.

Work training and social training are geared to the potentials of the patient, but always with empathy prodding. In most patients, progress is orderly, depending upon background abilities, profoundness of psychosis, family support, and to some extent, the length of hospitalization. However, we have seen patients follow Lloyd Douglas' description of the progress of society by climbing up two steps and falling back one. When this happens, the staff goes into as much detail as possible in determining the cause. Occasionally, the cause apparently lies in giving the patient more challenge than he can digest, but most often, the conclusion would suggest that something outside of the hospital rehabilitation environment was at fault.

A case history will illustrate this point:

A patient from a Central Virginia community had been hospitalized for about a year with a moderately severe schizophrenia with depression. Social history developed that he possessed an excellent work history, was well thought of in his community, was unmarried, and had devoted his mature life in caring for his mother and two sisters. In CHAP House, he progressed rapidly both in work and social endeavors. Drug therapy was discontinued; occasional psychotherapy appeared to be all that was needed. However, as he approached the furlough date and a job was secured for him in his local community, he regressed rapidly, with even some signs of suicidal content. Drug therapy was reinitiated and psychotherapy was increased. Finally, this patient was able to tell his physician that his thoughts of returning home brought up in him the recall of his terrific feelings of inadequacy in providing well for his mother and sisters. With much support from his psychiatrist and others, he was finally able to digest these feelings of inadequacy, return home, and is now carrying on a full-time job.

Social restoration is often the key to

community adjustment and is therefore given a large place in the CHAP House program. As we have mentioned, CHAP House is on the hospital grounds, subject to hospital regulations and served by hospital nurses and physicians.

It would seem that transfer to this facility would be without anxiety; yet, when one watches the new patients come in, they react by expressing their anxieties as do normals, with overactivity, underactivity, or withdrawal. Rarely do they take this transition "in their stride".

A recreational program, largely patient-inspired and directed, is a part of the CHAP program. The recreational therapist and staff give guidance and do arrange for all off-hospital ground activities; these are usually of the spectator sports variety. On-ground activities, such as socials, bazaars, plays, dances, and bingo parties are patient-organized and run to a large extent.

Open forums, or discussional periods, on such subjects as opening of a bank account, how to save money, the importance of appearance in seeking a job, the importance of work habits in staying on a job, what to do in case you are in an automobile accident, and the like—subjects which appear simple to us, but are of most importance to those who have lost contact with the day-to-day world of living. A simple visit downtown for the purpose of shopping or sightseeing raises such questions as "What will it cost?", "Whom do I pay?", "Where do I get off?", "How do I get back?", "Will those people look at me and think that I'm nuts?" These questions are usually answered by fellow patients, verbally and by guidance. From 30 to 40 patients visit downtown Petersburg every weekend. Forty-five patients went home on furlough during the past Christmas holidays, most of them going by normal transportation.

CHAP House rules and regulations follow those of the total hospital. They are not stringent, but necessary for smooth operation. As a guiding principle, the patient is informed on arrival that just because he has had a psychosis or in some other manner developed bad habits of thought, this does not give him the right to act in an unacceptable manner. Minor infringements are handled by the Patient Council; usually consultation alone is all that is necessary. Major infringements are handled by the staff and there have been surprisingly few major infringements. We have arrived at the conclusion that if we expect normality of our patients, they will usually live up to these expectations. A few patients have become actively psychotic, requiring return to the hospital ward for a period of treatment to return as they recover.

In summary, the restorative process in the field of the mentally and emotionally disabled is based on the principle of creating as normal an environment as possible of the work-a-day world and the live-a-day world, then assisting the patient in adjusting to same. Our first 18-month experience with our rehabilitation endeavors at Central State Hospital has been most rewarding.

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Diagnostic Laboratory Medicine . . . .

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Phenylketonuria—Phenylpyruvic Oligophrenia

The disorder phenylketonuria (PKU) is inherited as a Mendelian recessive characteristic and afflicts about four persons per 100,000. As an aid to premarital genetic counseling, the carrier state (heterozygote) can be identified by utilizing the phenylalanine loading test. Surveys of mental institutions originally suggested an incidence of about one in 40,000 persons, but intensive case-finding techniques have brought this figure near to one in 10,000 to 20,000. It may be even lower.

It is generally agreed that if phenylketonuria is detected early in life and if treatment to restrict phenylalanine intake is undertaken promptly, the severe mental retardation usually associated with phenylketonuria can be avoided.

The American Academy of Pediatrics' Committee on Fetus and Newborn recently recommended that a blood test for elevated phenylalanine be performed for all newborn infants prior to hospital discharge, and the adoption by many states of laws requiring testing of all newborns for phenylketonuria has created a problem of detecting this disease during the first few days of life. Experience has shown that if the diagnosis of PKU is not made as a result of routine screening during the newborn period or immediately following, it is unlikely that any test for phenylketonuria will be repeated until there is evidence of developmental retardation at six or nine months of age.

The problem is that infants with PKU appear to be normal at birth and during the neonatal period. Plasma phenylalanine levels are normal at delivery (normal values are 0.4 to 2 mg per 100 ml.) and phenylpyruvic acid does not appear in the urine until plasma phenylalanine levels rise to about 15 mg per 100 ml. This may occur as early as six days and as late as thirty-five days after birth. The plasma phenylalanine and the urine phenylpyruvic acid are completely dependent upon protein intake.

As a rule, increased levels of phenylalanine in the blood are associated with a mental defect. There are, however, documented instances in which persons with persistently excessive levels of phenylalanine in the blood have had normal intelligence. Conversely, mental retardation occurs in persons in phenylketonuric sibships who do not have phenylketonuria. The birth of mentally retarded children without phenylketonuria to a phenylketonuric mother suggests that cerebral damage may be caused in utero by placental transfer of increased amounts of phenylalanine from the maternal circulation.

In 1937 Penrose and Quastel introduced a method for quantitative determination of phenylpyruvic acid in urine based on the green color reaction with ferric chloride. The test can now be performed directly on a diaper or a piece of filter paper can be placed on a diaper; urine dried on filter paper is stable for several months. Ames markets a reagent impregnated paper strip called Phenistix,® which turns gray to gray-green when positive for phenylpyruvic acid. Results obtained with Phenistix® compare satisfactorily with the liquid ferric chloride test.

A screening test for the detection of an abnormal level of serum phenylalanine (the bacterial inhibition assay method of Guthrie) is being widely performed after the fourth to sixth day of life and not until the infant has had dietary protein (mother's milk for at least 72 hours). More recently the fluorometric method of McCaman and

Robins has also received wide application. Hill and associates have successfully automated the fluorometric method for the analysis of serum phenylalanine.

Mass screening programs based on tests for elevation of blood phenylalanine concentrations have revealed a large number of infants in whom the elevation of phenylalanine cannot be attributed to classical phenylketonuria. A more complete definition, then, must be applied to describe the disease condition which both requires and responds favorably to treatment with the phenylalanine-restricted diets. Since serious questions have been raised as to the effectiveness of early treatment in preventing mental retardation associated with phenylketonuria, a more definitive diagnosis is necessary in order to accumulate data related to treatment. If benefit is to be derived from legislation already passed in many states, requiring testing of infants for phenylketonuria, it is essential that the diagnosis be made properly as well as promptly.

Until recent years the diagnosis of phenylketonuria was based on the initial observation of a mentally retarded child who was found to have a positive ferric chloride reaction in the urine and an elevated serum level of phenylalanine. The sequence of events has been altered by the development of routine screening programs for phenylalaninemia in the newborn infant, which have led to an increased finding of high serum phenylalanine levels during the neonatal period. An approximate estimate is that 1:500 newborn infants have levels greater than 4 mg per cent. It has become increasingly apparent that not all these infants with "phenylalaninemia" have phenylketonuria in the classical sense. At present there is evidence for at least three types of "phenylalaninemia" as determined by the screening tests. These types are characterized as follows:

1. The newborn infant with classical or true phenylketonuria is characterized by rapid rise in serum phenylalanine during the first 10 days to levels greater than 30 mg per cent, increased concentrations of phenylalanine, orthohydroxyphenylacetic acid and phenylpyruvic acid in the urine, a "mousy odor" associated with phenylacetic acid in the urine and sweat, and a low or normal serum tyrosine level. Characteristic clinical and neurological findings usually cannot be detected in the newborn period. Early clinical evidence of neurological involvement is suggested by a decreased placing response and fall-away reaction, persistent flexion tone, and the presence of low threshold reflexes. Electroencephalograph may, on occasion, reveal spike discharges. Phenylalanine loading tests will usually show both parents to be carriers.
2. The second group is characterized by a serum phenylalanine concentration usually less than 15 mg per cent, and tyrosine levels ranging from 10 to 75 mg per cent. It includes about 25 per cent of low birth weight infants, but also includes some full-term infants of normal weight. The pathogenesis is best explained by delayed maturation of the phenylalanine hydroxylase and parahydroxyphenylpyruvic oxidase systems. There is excretion of parahydroxyphenylpyruvic acid in the urine. Administration of 100 to 200 mg vitamin C daily will usually produce a rapid fall in both serum tyrosine and serum phenylalanine levels.
3. This newborn infant group has a slower rise in the serum phenylalanine concentration to levels of less than 30 mg per cent, and normal serum tyrosine values. Phenylpyruvic and orthohydroxyphenylacetic acid sometimes may be found in increased amounts in the urine. The serum phenylalanine level may remain elevated for considerable periods of time, but at lower levels than in "true" phenylketonuria.

The nature of the biochemical defect is not known; quite likely this group consists of more than one entity. Whereas in the first group the frequency with which siblings are found to have phenylketonuria is consistent with inheritance of an autosomal recessive trait, in this group the frequency of classical phenylketonuria in siblings is quite low.

Undoubtedly, there is overlapping between these groups. Group 1, classical phenylketonuric patients, is probably the best defined; Group 3 is the least well defined. The information available, however, points to the fact that the syndrome "phenylalaninemia" is still poorly defined and that there is great need for better clinical, biochemical, and genetic differentiation of the entities which cause an increased serum phenylalanine in the infant during the newborn period and subsequently.

A genetic interpretation for the clinical and biochemical variations in typical phenylketonuria is based on three levels of genetic heterogeneity: 1. a genetic defect at the primary locus leading to an abnormality of enzyme activity; 2. genetic differences in secondary metabolic pathways leading to a range of biochemical abnormalities, and 3. genetic differences in the response of the central nervous system to different substances producing a range of neurological differences. Thus, genetically determined differences between affected persons in secondary metabolic pathways and central nervous system responses may lead to considerable phenotypic variation. This interpretation may serve to explain untreated phenylketonuric individuals who have normal intelligence; it may also explain some of the variation noted in the severity of the biochemical abnormalities in the newborn infant with "phenylalaninemia".

In order to assure that protein intake is sufficient to yield an elevated blood phenylalanine concentration in the phenylketonuric infant, the Committee on Fetus and Newborn of the American Academy of Pediatrics has made the following recommendation: "A blood test for elevated concentration of phenylalanine performed no sooner than 24 hours after onset of milk feeding and prior to discharge is recommended for newborn infants."

It has been reported that concentrations of blood phenylalanine of less than 4 mg per 100 ml. are not significant.

The assumption is generally made that phenylalanine or one of its metabolites is toxic to the developing brain. As yet there is no evidence to support this statement. Animal studies have been inconclusive.

Summary

New methods are mentioned for rapid and economical screening of large numbers of hospital nursery infants for elevation in blood phenylalanine associated with phenylketonuria. Problems, still unsolved, are the uncertain diagnosis of phenylketonuria and the need for improved clinical, genetic, biochemical, and possibly psychological definition of "phenylalaninemia".

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Public Health . . .

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Rheumatic Fever Prevention—1968

The incidence of rheumatic fever has dropped precipitously in the last 30 years. Two responsible factors are the introduction of antibiotic therapy and the general improvement in living conditions throughout the world. Understanding the relationship of the beta hemolytic streptococcus to the initiation of the disease, as well as to the prevention of recurrences, has been specifically helpful. Large surveys conducted between 1948 and 1960 in school children showed an incidence of rheumatic fever between one and two per thousand. In the college population, the rate is about eight per thousand. In Virginia, there is no comprehensive statewide register of new rheumatic fever patients. Six state pediatric cardiology clinics recorded 286 new cases of rheumatic fever last year. Two of the 53 patients admitted to the Medical College of Virginia Hospital in 1967 died of fulminating carditis. Rheumatic heart disease is still the greatest killer from heart disease under 40 years of age and deserves continuing attention in both primary and secondary prophylaxis.

Primary Prevention

It is now firmly established that rheumatic fever cannot be initiated without the occurrence of group A beta hemolytic streptococcal infection. This organism is found universally throughout the world and in all seasons, although it is more prevalent in northern climates and in the winter and spring months. Some streptococci are more virulent than others. School children will average approximately one streptococcal infection every two years. If not adequately treated, streptococcal infections result in rheumatic fever in 0.3-3.0 percent of cases: the higher incidence being quoted from epidemics in the military service. Recent studies from Boston on patients admitted with acute rheumatic fever showed that none had been adequately treated for their initial streptococcal infection, if it had been recognized at all. About one-third of streptococcal infections are silent, but a diagnosis could be made in the others. No beta hemolytic streptococcus has yet been demonstrated to be insensitive to penicillin and 10 days of this bactericidal drug will eradicate the organism in more than 95 percent of cases.

What are the manifestations of streptococcal infections in the child under five, the early school child and in the older patient? They vary as demonstrated in Table I. The child under four years with less

TABLE 1

CLINICAL DIAGNOSIS OF STREPTOCOCCAL INFECTIONS

Ages	*Suggests Strep*	*Probably Viral*
Less than 4 years	Prolonged coryza. Draining ears. Impetigo.	Croup. Cough. Wheeze.
4-8 Years	Scarlet rash. Sore throat— beefy red. Petechiae on palate. High temperature. Coryza with purulent complications.	Cough. Hoarseness. Vesicles on palate.
Greater than 8 Years	Very sore throat. Beefy red tonsils. High temperature. Large, tender cervical nodes. Petechiae on palate. WBC greater than 12,000 chiefly polyps.	Coryza. Low or no temperature. Cough. Vesicles on palate. WBC less than 10,000, chiefly lymphs.

This article was prepared by Dr. Carolyn M. McCue, Director, Pediatric Cardiology, Medical College of Virginia in her capacity as Director, Children's Cardiology Program, Bureau of Crippled Children, Virginia State Department of Health.

hyperimmune reaction to the streptococcus is more prone to the protracted course of coryza and purulent complications. The child from four to eight years is much more likely to get the sensitization reaction such as rheumatic fever or nephritis. He may manifest the first stage by a scarlet rash, impetigo, protracted coryza or the more fulminating sore throat. The typical features seen in older children or adults of acutely sore throats with petechiae on the palate can be diagnosed correctly clinically even by experts only in 75 percent of patients. Our goal is to make the streptococcal diagnosis early and prevent rheumatic fever.

Laboratory confirmation is essential and requires one of the following:

1. A positive throat culture for beta hemolytic streptococcus, group A.
2. An antistreptolysin titre elevated to greater than 250 Todd units.
3. A white count greater than 12,000, chiefly polymorphonuclear cells, is highly suggestive if the clinical findings are as indicated in Table I.

Throat cultures are available in every major hospital in Virginia and without charge in 10 Health Department Laboratories. Antistreptococcal titres are also available in most hospitals and in three Health Department Laboratories.

Once the diagnosis of a beta hemolytic streptococcal infection is made, how should this be treated in the general population? Penicillin is bactericidal and highly effective if given early, in adequate dosage and for at least 10 days. The American Heart Association has given specific directions and recommendations as noted in Table II.*

Who Should Be Treated

1. Patients with clinical signs and symptoms as listed in Table I, who have a positive culture for beta hemolytic streptococcus or an elevated streptococcal titre or scarlet fever rash.
2. Patients with a diagnosis of acute rheumatic fever according to Jones Criteria. Those with polyarthritis or chorea must be given this initial treatment as well as those with heart disease.

TABLE II

RECOMMENDED TREATMENT SCHEDULES FOR STREPTOCOCCAL INFECTIONS

Intramuscular Penicillin
Benzathine penicillin G

Children—one intramuscular injection of 600,000 to 900,000 units. The larger dose is probably preferable for children over 10 years of age.

Adults—one intramuscular injection of 900,000 to 1,200,000 units.

Oral Penicillin

Children and adults—200,000 or 250,000 units three or four times a day for a full 10 days. Therapy must be continued for the entire 10 days even though the temperature returns to normal and the patient is asymptomatic.

Combined Therapy

Various combinations of oral and intramuscular penicillin should be effective provided adequate coverage is continued for 10 days.

Other Antibiotics

Erythromycin is the most effective antibiotic in patients who are sensitive to penicillin if given for 10 days.

Not Recommended

The Sulfonamide drugs should not be used for the treatment of streptococcal infections. In an established infection, they will not eradicate the streptococcus and therefore will not prevent rheumatic fever.

3. Siblings or close associates of new patients with acute rheumatic fever with minor symptoms or a positive throat culture.
4. In an epidemic of beta hemolytic streptococcus, institutionalized children in an orphanage or members of a military installation may require mass prophylaxis.
5. Asymptomatic carriers of beta streptococci found on routine cultures are not recommended for therapy.

It should be the prime responsibility of all physicians in Virginia, when seeing patients with suspected streptococcal infections, to make adequate tests which confirm

or deny the diagnosis and to treat adequately for 10 days all patients in whom the diagnosis is suggested clinically or in the laboratory. Only in this way can the primary or initial prevention of rheumatic fever occur with greatest efficiency. Is it not far better to prevent a case of rheumatic fever and heart disease than to treat it later?

Secondary Prevention

The diagnosis of rheumatic fever remains a clinical and often complex one. The following plan does not apply to all suspicious cases, but only to those with a firm diagnosis according to Jones Criteria.* The most striking characteristic of rheumatic fever is the tendency to recurrence, which is greater in younger patients, in those with heart disease and in the first year or two after the initial episode. While recurrence rates prior to prophylactic therapy were over 60 percent, adequate prophylaxis can reduce this to about 3 percent with oral drugs or less than 1 percent with injectable benzathine penicillin. Since heart disease increases with recurrences, once an individual has shown his susceptibility to the streptococcus, every effort should be made to avoid another infection. The American Heart Association has given specific directions for secondary prevention as in Table III.*

TABLE III

PROPHYLAXIS OF RHEUMATIC FEVER IN RHEUMATIC FEVER PATIENTS

Benzathine Penicillin G Intramuscular	1,200,000 units every 28 days.
Sulfadiazine—Oral	1 Gm. once a day for patients over 60 pounds: 0.5 Gm. for patients under 60 pounds.
Penicillin—Oral	200,000 or 250,000 units twice a day. Twice daily is more effective. Although other kinds of penicillin may be used, buffered penicillin G is satisfactory.

Bicillin is currently used for patients with the greater tendency to recurrence, notably those in the younger age group and those who have more severe varieties of heart disease. Allergic reactions are rare. This method is the preferred one for at least a year or two after the initial attack. Even conscientious patients sometimes forget oral penicillin or sulfadiazine, which are about equally effective. The duration of therapy for patients with heart disease is indefinite. Selected children over 16 years of age who had a single attack more than five years previously and who have no residual heart disdisease are being discontinued from their prophylatic regimes under careful orbservation. Preliminary studies in New York show their recurrence rate to be extremely low.

Problems of maintaining prophylaxis over a prolonged period in many patients who have the diagnosis of rheumatic fever is medically, emotionally and economically difficult. Registers need to be more complete in our State and hopefuly will be instituted in the near future. The six Rheumatic Fever Clinics throughout the State work with the Public Health Departments of the counties to supply prophylactic drugs free, or at minimal cost, to indigent patients who are being followed in the State clinics. Education in continuing need for this therapy is a duty of physicians directing the clinics, as well as referring doctors and nurses. Adolescents pose a major challenge. Recurrences tend to mimic the initial episode of rheumatic fever in major manifestations. Hence, heart disease is more likely to worsen in a child who had initial heart disease. But even a diagnosis of chorea or polyarthritis cannot be ignored because about 10 percent of such patients develop heart disease with subsequent exacerbations. Long term faithful prophylaxis is a sound investment, since it is the most efficient method known for avoiding the need for cardiac surgery for mitral stenosis or severe aortic valve disease later.

Prevention of Bacterial Endocarditis

A final responsibility of physicians caring for patients with residual heart disease,

either of rheumatic or congenital origin, is the prevention of bacterial endocarditis by the administration of appropriate drugs preoperatively when surgery is contemplated around the mouth and nose. A dental extraction or tonsillectomy has a high incidence of bacteremia. Bacteria may lodge on damaged valves and form vegetations. Cardiac surgeons recognize that the most severely deformed valves requiring replacement, initially suffered rheumatic or congenital defects but were damaged more severely by bacterial involvement, in spite of intensive therapy. Both patients, their physicians and dentists should be educated to take the precautions as suggested by the American Heart Association as in Table IV.*

TABLE IV

PROPHYLAXIS FOR PREVENTION OF BACTERIAL ENDOCARDITIS

Day of Procedure:

Procaine penicillin 600,000 units supplemented by 600,000 units crystalline penicillin intramuscularly one to two hours before procedure.

For Two Days After Procedure:

Procaine penicillin 600,000 units intramuscularly each day. In selected instances, 0.25 Gm. of alpha-phenoxymethyly penicillin (Penicillin V) or 0.25 Gm. of alpha-phenoxyethyly penicillin (phenethicillin), or 500,000 units of buffered penicillin G four times daily by mouth on each day may be used for those patients in whom full cooperation is anticipated and ingestion is assured.

Contra-indication to Above:

The main contra-indication is sensitivity to penicillin. Under such circumstances, erythromycin should be used in a dose of 250 mg. by mouth four times a day for adults and older children.

Conclusion

Every physician in 1968 treating children or adults has a responsibility to consider the diagnosis of streptococcal infections in patients he encounters. He must know the clinical manifestations at different ages, the laboratory facilities available in his area and the appropriate treatment for an infection once it occurs. The dissemination of this information to physicians throughout the world is largely responsible for the reduction in rheumatic fever and rheumatic heart disease, which can be primarily prevented whenever overt streptococcal infections are recognized and treated.

Once the manifestations of acute rheumatic fever have occurred, it is the physician's responsibility to make a firm diagnosis as soon as possible. Long term prophylaxis against recurrences will minimize further heart damage and should be continued as recommended by the American Heart Association. Hopefully, a register for patients with rheumatic fever and rheumatic heart disease in Virginia can be established within the next few years. Only in this way can we evaluate our current program and plan more effectively to meet our needs in the future.

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*The following pamphlets may be obtained from your local Heart Association or the American Heart Association:

"Prevention of Rheumatic Fever"

"Jones Criteria for Guidance in the Diagnosis of Rheumatic Fever"

"Prevention of Bacterial Endocarditis"

MONTHLY REPORT OF BUREAU OF COMMUNICABLE DISEASE CONTROL

	Feb. 1968	Feb. 1967	Jan.-Feb. 1968	Jan.-Feb. 1967
Brucellosis	0	5	0	9
Diphtheria	0	0	0	0
Hepatitis	34	59	78	107
Meningitis (Aseptic)	0	1	0	2
Meningococcal Infections	6	5	12	10
Poliomyelitis	0	0	0	0
Rocky Mt. Spotted Fever	0	0	2	0
Rubella	85	41	105	59
Rubeola	39	228	58	427
Streptococcal Sore Throat (including Scarlet Fever)	1543	1572	3110	3149
Tularemia	0	0	0	0
Typhoid Fever	1	1	1	1
Rabies (in animals)	19	16	36	32
Venereal Diseases				
Syphilis	119	162	295	332
Gonorrhea	938	777	1811	1451
Other	3	0	3	0

Cancer Trends . . . .

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The Question of Stilbestrol in the Treatment of Prostatic Carcinoma

Truth is never pure, and rarely simple.[1]

Some years ago the Veterans Administration formed a cooperative group of urologists to study certain aspects of prostatic carcinoma. Recently, some of their results have been published[2,3] and portions of their conclusions quoted, often out of context, in various local and national meetings and in professional and semi-professional informational media. The results of these references to the complete, published data has often left more confusion than enlightenment. The most often cited result of the group study is to the effect that stilbestrol is of no value in treating patients with prostatic carcinoma and may be contraindicated, since the stilbestrol-treated patients did not survive as well as those treated with a placebo. Further, the causes of death in this group of treated patients were likely to be vascular rather than neoplastic in nature.

All of this is quite paradoxical since nearly all physicians feel that most patients with advanced prostatic carcinoma experience a remission when given stilbestrol. Huggins (now a Nobel Laureate for the role he played in these studies) and Hodges[4] first reported on their observations in 1941 and it seems scarcely possible that the hundreds of confirming reports could have been prepared by either idiots and/or malicious quacks. Surely, it couldn't have taken twenty-five years for an intelligent, honest man to test the thesis and find it wanting. What, then, are the special circumstances that might allow both positions to be equally true?

First of all, the term, "prostatic carcinoma", envelops a mass of different kinds of tumors, many with different biological potentials. As a group they are heterogeneous and display this characteristic by behaving in a capricious fashion.

For instance, Greene and Simon[5] studied a group of patients with symptoms and findings of benign prostatic hyperplasia and in whom well organized, unsuspected carcinoma was found. Whether treated with stilbestrol or not, the survival rate approached that of the population at large. In an analysis of a very large population of patients also treated at the Mayo Clinic, Emmett and his associates[6] showed that grade exerted a powerful influence on survival in hormonally treated patients; the lower the grade the longer the survival. There is also the strong influence of stage on survival. If Stage A is found in patients with no manifestation of the disease and in whom it can be found only on microscopic examination of the prostate, Stage B is the isolated prostatic nodule with no other manifestation of the disease, Stage C is clinically manifest by virtue of urinary symptoms of prostatism and, on examination, by extension of the neoplasm to the seminal vesicles, base of the bladder and lateral pelvic walls in varying degrees and Stage D is clinically manifest by extension to soft tissues and bones and/or hydronephrosis on the basis of ureteral obstruction, then it can be shown

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that the patients found in Stage A survive very well in terms of five years if they are untreated by any modality. On the other hand, about 80% of the patients in Stage D are dead five years from the beginning of any kind of treatment, and, these patients are dead, largely, of carcinoma. Obviously, then, one must know a lot about the stages of prostatic carcinoma if one is to judge the results of therapeutic efforts. Next, one must accept the truth that few statistically sound studies on any clinical disorder are available and this includes prostatic carcinoma, though the most inclusive effort is that of the VA Group. They separated their patients by stage in a fashion roughly similar to that described above. Then they assigned the patients on a random basis to a variety of therapeutic regimens. The treatment assigned was placebo (yes, placebo), stilbestrol, 5 mg. daily, orchiectomy, orchiectomy plus placebo and stilbestrol plus orchiectomy. Those patients in stages A & B also underwent radical prostatectomy for purposes of this study. Analysis of the survival data demonstrate that over half of all deaths were attributed to disorders other than prostatic carcinoma. While patients who did not receive stilbestrol had a higher mortality from prostatic carcinoma than those who did, the patients who received stilbestrol had a higher percentage of deaths from other causes. In short, stilbestrol reduced the prostatic carcinoma rates but these patients had an increase in total mortality from non-cancer causes. Further, this seems to be a true acceleration of non-cancer mortality and not the result of reducing deaths from cancer.

Thus, it appears that in patients with prostatic cancer, stilbestrol is associated with an increase in lethal cardiovascular disease. However, this study falls far short of statistical impeccability. For instance, in the published text there is no statement about the number of patients lost to follow-up nor is there one about the success of follow-up, and 26% of the number of patients admitted to the cooperating institutions were not admitted to *any* treatment group because of poor general condition and/or other items. Another 10% were excluded after admission to the study because they did not meet the criteria of acceptance and another 27% of those admitted were changed from the study protocol so that another form of treatment could be administered. Thus, one receives the impression that many patients with progressive lethal carcinoma—bad cancer—were excluded initially. Another large segment of patients with progressive carcinoma had their treatment altered, quite likely from placebo to hormonal therapy of some variety.

At this point, one can reach a certain rapprochement with the conflicting expression of opinions. *When placebo-treated patients in the VA Study had progressive disease and were then treated by one of the hormone protocols, over 60% showed an arrest in the progression of the disease and over 40% demonstrated definite clinical improvement.*

Therefore, it appears that identification of the kind of prostatic carcinoma in each individual is essential. Low-grade, low stage disease is probably best left untreated by any modality while progressive prostatic carcinoma which is productive of symptoms and disability can be best treated by orchiectomy. A decision regarding the exact status of stilbestrol therapy probably must await careful biological studies designed to determine the dosage required to ablate endogenous testosterone production and remission of the neoplastic process.

Obviously, the study raises as many questions as it answers and should prompt other investigators to re-examine the proposal that estrogen therapy is of value in the treatment of arteriosclerosis, cardiovascular disease and menopause.

Editors' Comments

Many of us have been confused and perplexed by recent reports that seem to ques-

tion our time honored concepts of the hormonal control of prostatic cancer. Dr. George R. Prout, Jr., of M.C.V. has been kind enough to give us his own opinions of this issue. Dr. Prout is a member of the Task Force that advises the National Cancer Institute concerning problems relating to research in urologic oncology. With this background his concepts and questions are certainly authoritative and worthy considerations.

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Federal Income Taxes—(Health Insurance Premiums and Benefits)

Health Care Insurance—Prior to 1967, Federal Income Tax Law provided for the inclusion of health insurance premiums paid by the taxpayer in calculating the medical expense deduction. Generally, the deduction was limited to the amount by which medical care expenses exceeded 3% of adjusted gross income (with a special 1% deduction applicable to the cost of drugs and medicines), although expenses for persons over 65 were not subject to the 3% rule.

While the Internal Revenue Service never acquiesced, court decisions in several cases permitted inclusion of premiums for loss of time and accidental death and dismemberment benefits with premiums for medical care insurance.

Under the new law effective January 1, 1967, a special deduction is allowed for premiums for insurance covering the cost of medical care, but premiums for loss of time and accidental death and dismemberment benefits may not be included. The new medical care deduction is the sum of:

(1) An amount (not to exceed $150) equal to one-half of the premiums paid during the taxable year for medical care insurance for the taxpayer, spouse and dependents. The other half of the premiums for medical care insurance, plus any excess of the first half over the $150 limit, continues to be deductible as explained under (2) below; and

(2) The amount by which expenses paid during the taxable year for medical care for the taxpayer, spouse and dependents (including medical care insurance premiums in excess of the $150 deductible under (1) above) exceed 3% of his adjusted gross income (subject to the same 1% limitation applicable to drugs and medicines under the present law). Where medical insurance is included in a contract with other benefits, the medical care premium must be separately stated in the contract, or available in a statement from the insurance company. Benefits paids as reimbursement for medical care expenses are not taxable income.

Loss of Time Coverage—Premium paid by the taxpayer for disability income are not deductible, except Business Overhead Expense insurance, the premiums for which are deductible as business expense. However, benefits for loss of time received under policies for which the taxpayer pays the entire premium are not taxable income. The rules applicable to benefits under plans for which an employer pays all or part of the premium remain unchanged.

The Medical Society of Virginia . . . .

Minutes of Council

A meeting of the Council of The Medical Society of Virginia was held on Sunday, February 11, at Society headquarters.

Members Present: Dr. Thomas W. Murrell, Jr., Dr. F. Ashton Carmines, Dr. K. K. Wallace, Dr. W. Leonard Weyl, Dr. Mack I. Shanholtz, Dr. Harry J. Warthen, Dr. W. Callier Salley, Dr. Raymond S. Brown, Dr. William S. Hotchkiss, Dr. William R. Hill, Dr. William Grossmann, Dr. F. H. McGovern, Dr. Harry B. Stone, Jr., Dr. William D. Liddle, Dr. Carl E. Stark, and Dr. Carl P. Parker, Jr.

Others Present: Dr. Dennis P. McCarty, Dr. W. Linwood Ball, Dr. Allen Barker, Dr. Hunter McGuire, Jr., Mr. William Miller and Mr. Gerald W. Blanchard.

Tetanus

Dr. William Sandusky, Charlottesville, represented the Trauma Committee of the Virginia Chapter, American College of Surgeons, and reported that there are approximately 1.5 million residents of Virginia who have not been immunized for tetanus. The Trauma Committe is most anxious that mass immunization campaigns be conducted over the State and has arranged a meeting with representatives of component societies for the purpose of exploring such a possibility. While the Trauma Committee would provide as much guidance as possible, all work would be done by physicians at the local level.

Dr. Sandusky stated that two such campaigns had already been held in the State and proved quite successful. In Norfolk some 75,000 persons have been immunized while Lexington can point to an 11,000 figure. He explained that much of the promotion can be handled by the Department of Health and that an individual contribution of 50¢ is usually more than enough to pay for the toxoid.

Dr. Shanholtz spoke on behalf of the Department of Health and indicated that the Department would be happy to assist with promotion and publicity, obtaining clinic station locations, etc. First, however, he wanted to make sure that such campaigns had the full approval of The Medical Society of Virginia.

There followed considerable discussion concerning problems almost certain to arise and a question was raised as to just where preventive medicine of a public health nature actually leaves off and the private practice of medicine begins. It was agreed that both children and adults would be eligible, although answers to certain questions were needed from the Virginia Pediatric Society.

The question of professional liability was also discussed and it was agreed that physicians participating should make sure that they are adequately covered by their insurance. It was also proposed that all those receiving the toxoid should give written permission.

A motion by Dr. Hotchkiss that the proposed immunization campaign be endorsed by The Medical Society of Virginia was seconded and adopted.

Health Survey

Dr. Murrell briefly reviewed those events leading to the proposal for a comprehensive survey of health services in Virginia and stressed the importance of involvement by physicians in activities of this kind. He expressed the feeling that it has never been more important for physicians to conduct their own studies and do their own planning. He reported that a Special Committee had been appointed to meet with representatives of the Department of Health, Education and Welfare in an effort to develop a plan of action acceptable to both the Society and HEW.

Dr. Birdsong, Chairman of the Special Committee, then reported on meetings with HEW officials. He stated that the Society's original proposal was considered too lengthy and it was necessary to decide on two or three specific objectives. Application would then be made for a planning grant which hopefully would lead to an operational grant. Funding would be on a contract rather than a grant basis. This would eliminate a great deal of preliminary work and permit the necessary funds to be approved in Charlottesville.

Dr. Birdsong went on to present three examples of specific objectives:

A. Two phase evaluation of physician and patient reactions to delivery of health services;

B. The role of, and need for, additional and better

trained paramedical or allied health professionals; and,

C. A definition of the "gray zone" of medical need.

A question was raised as to who would actually have control of the survey and ultimate disposition of survey findings. Council was assured that The Medical Society of Virginia would be in complete control and would make all decisions concerning release of material, etc. It was recognized that staffing such a survey would be quite a problem, but that the Society would be free to employ whomever it might wish.

It was generally agreed that objectives "B" and "C" had much to recommend them. Objective "A" was not liked quite as much but its public relations potential was recognized. Dr. Birdsong expressed the feeling that he was now in a position to go back and have his Committee pull all the pieces together into one proposal.

Dr. Weyl then moved that objective "B" be selected as the initial project for the survey and that it serve as a platform upon which to incorporate parts of "A" and "C". The final proposal would be submitted to the President for distribution to Council. *The motion was seconded and adopted.*

Regional Medical Program (Heart, Cancer, Stroke)

Dr. Murrell reported late developments in the Regional Medical Program and stated that emphasis would be placed on continuing education. He advised Council that The Medical Society of Virginia would be one of the grantees—along with the two medical schools, the Virginia Hospital Association and Norfolk Medical Authority.

In line with Dr. Murrell's feeling that physicians should involve themselves with the various programs, the Heart, Cancer, Stroke Committee of The Medical Society of Virginia has been designated the Project Review Committee of the Regional Medical Program. This particular Committee would review all proposed projects and recommend acceptance or rejection to the Governor's Advisory Committee. This is somewhat similar to the procedure followed in North Carolina and certain other states. Dr. Murrell stressed the fact that the Society, under this arrangement, has a real responsibility to fulfill.

Virginia Association of Professions

Dr. Liddle, a member of the VAP Board of Directors, reported that this young organization has great potential. He pointed out that medicine needs allies and that VAP brings together professional people who share a common philosophy. The Virginia Association of Professions now has a Legislative Committee and is trying hard to develop an educational program.

Dr. Liddle went on to state that physician membership in VAP is much too small and that ways and means must be found to increase the total. He reported on a suggestion by the VAP that consideration be given to including that organization on the annual statement of The Medical Society of Virginia —with a voluntary contribution of $4.00. It was agreed that VAP has much to recommend it. It was also pointed out that The Medical Society of Virginia is a charter member and pays annual organizational dues of $200. Some fear was expressed that the Society's dues statement could very easily become overloaded. A voluntary contribution for VaMPAC is already included.

A motion by Dr. Liddle that the Society take the lead in setting up a mechanism for developing dual membership on a voluntary basis with dues not to exceed $4.00 was lost for want of a second.

Congressional Luncheon

Everyone agreed that the annual Congressional luncheon should be continued and that last year's format be followed once again. This means that members of the Committee on National Legislation will go directly to the offices of their Congressmen and visit with them before escorting them to the luncheon. A concise report on key issues before Congress will be prepared by AMA and placed in the hands of all Committee members before they leave home. Thus, there would be no meeting in the Washington office of AMA.

It was then moved by Dr. Hill that the luncheon be held again this year. *The motion was seconded and adopted.*

It was further agreed that the Executive Secretary be permitted to establish the date after conferring with members of Virginia's Congressional delegation.

Belroi

Dr. Murrell stated that for many years Dr. Brown and his three-man Walter Reed Committee have

done a wonderful job of supervising the maintenance of "Belroi", the Walter Reed birthplace. Dr. Brown and Committee members have made sure that the grounds are always presentable and ready for visitors. This has been a big job and much thought has been given over the years to finding a reputable organization which would be interested in accepting Belroi as a gift and thus assuming responsibility for its maintenance.

The Association for the Preservation of Virginia Antiquities has been approached by Dr. Murrell in this regard and has indicated its willingness to accept the birthplace as a gift. It has also indicated that continued financial assistance by The Medical Society of Virginia would be greatly appreciated and most helpful. The annual budget has included a $500 item for a number of years.

Dr. Brown stated that the Walter Reed Committee was in agreement that the APVA would be the perfect organization to take over the ownership and maintenance of Belroi. He then moved that the house and property be deeded to the Association for the Preservation of Virginia Antiquities and that a financial contribution towards maintenance of the property be included in the Society's annual budget. The motion was seconded.

During the ensuing discussion it was brought out that the Woman's Auxiliary was very much interested in finding articles of interest to place in Belroi. Dr. Brown and Dr. Murrell expressed the feeling that the APVA would work with the Auxiliary in every possible manner.

Council then adopted Dr. Brown's motion with the understanding that it was subject to final approval by the House of Delegates.

Attention was called to the fact that a study of papers attributed to Dr. Walter Reed is currently under way in Charlottesville and this might possibly be of interest to the Society's Program Committee for the Annual Meeting.

Legislative Report

Mr. Miller then reviewed those bills before the General Assembly which are of particular interest and importance to physicians. It was learned that the Legislative Committee had met on two occasions and Mr. Miller had appeared before a number of Committees of the General Assembly in order that the Society's position on the various pieces of legislation might be made known. Mr. Miller discussed in some detail those pieces of legislation sponsored by the Society. They include a bill seeking to resolve the problem of drug dispensing by physicians, a lay laboratory control bill, a proposed amendment to the sales tax statues, a bill to exempt members of utilization review committees from malpractice liability, etc.

Other bills in which the Society has a great interest are those having to do with the defining of blood services, glue sniffing, sales of syringes, supplemental funds for Hill-Burton, simplification of commitment procedures, certification of ambulance operators, admission of certain books, articles, etc. as evidence in malpractice cases, an over-all study of the Virginia Pharmacy Code, an extension of the Good Samaritan Act, amendments to the Hospital Authorities Act, nursing education, State Board of Medical Examiners, physical therapy assistants, medical examiners, etc.

Mr. Miller reported also that legislation had already been adopted permitting the use of studded snow tires during certain months of the year. This legislation carried an emergency provision and is now in effect.

Dr. Grossmann spoke in favor of an amendment to the Hospital Authorities Act which would permit practicing physicians to serve as members of the Hospital Authorities concerned. This is in keeping with resolutions on the subject adopted by both the American Medical Association and The Medical Society of Virginia. Hope was expressed that members of Council would contact members of the Committee of Counties, Cities and Towns who might be from their areas.

At this point Dr. Shanholtz commented on several bills of particular interest to the Department of Health. One of these (HB 555) originally required mandatory vaccinations and Dr. Shanholtz did not consider it enforceable. As a result of meetings with the patron of the bill, it has been rewritten in such manner that most objections have been removed. It was agreed that there was little reason to oppose it.

Dr. Shanholtz also discussed the responsibilities of his Department under the National Safety Act—particularly where training programs for rescue squads, etc. are concerned. A suggestion was made that the Virginia Chapter, American College of Surgeons, be contacted for advice and other assistance.

Consideration was then given a bill which would enlarge the State Board of Health from seven to

nine members and set individual terms at four years. Dr. Shanholtz indicated that this would merely bring the Board of Health in line with other State Boards. Although the two additional members could be either members of a profession or laymen, it was believed that at least one additional physician would be appointed. *A motion was then adopted requesting Dr. Murrell to urge the Governor to appoint at least one additional physician to the State Board of Health.*

Committee on Pharmacy

Council was advised that Virginia law provides for five members of The Medical Society of Virginia to be included on a special committee which reviews and recommends those drugs to be included under the Drug Abuse Act. Since the Society's five representatives also happen to be members of the Society's Committee on Pharmacy, a suggestion had been received that that particular Committee be designated a standing committee. This, of course, would require an amendment to the By-laws.

It was brought out during the discussion that the President can always make such appointments as he desires and, consequently, there is some question as to the value of adding another standing committee. No further action was taken.

Osteopathy

Although the Society's Special Committee on Osteopathy had met to consider the question of hospital privileges for osteopaths, its final report was not yet ready for consideration. Council will undoubtedly have the Committee's recommendations before it at its next meeting.

It was learned that a member of the Society had indicated a wish to associate with an osteopath and asked for some expression by Council. The same member also asked if there would be any objection to employing a podiatrist and/or optometrist.

It was brought out during the discussion which followed that AMA expressed the feeling that all policy matters having to do with osteopaths and physicians should be handled and decided at the state level. The thought was expressed that every effort must be made to avoid complications—particularly since osteopaths do not enjoy hospital privileges at the present time.

A motion was then introduced expressing the opinion that it is not appropriate at this time for a doctor of medicine to associate with a doctor of osteopathy. The motion was seconded and carried.

It was also agreed that this same policy should apply to the employment by a physician of a podiatrist and/or optometrist.

Blue Shield

Dr. Hill reported that the Blue Shield Commission had already held two meetings and that considerable progress had been made in solving problems surrounding the 7510 Plan of the Virginia Medical Service Association. It was the Commission's recommendation that the Plan be approved with the following three modifications:

1. Physicians' fees would be paid on a "usual and customary" basis.
2. Fees which seem to be in excess of usual and customary would first be reviewed by Blue Shield's own Schedule of Allowances Committee. Only those cases not approved for full payment by that Committee would be referred to a Special Review Committee of The Medical Society of Virginia.
3. Profiles developed through recent surveys would be returned to individual physicians. Present guidelines would be under constant review and revisions made as indicated from claims experience.

Dr. Hill then moved that the Commission's recommendation concerning the 7510 Plan be approved. The motion was seconded.

There followed considerable discussion during which Dr. McCarty voiced the fear that the Society might indeed be setting a precedent with respect to the elimination of income limits. Dr. Weyl was of the opinion that the words "billed fee" should be substituted for the words "usual and customary" in modification "1". This, in his opinion, would eliminate any confusion surrounding the words "usual and customary"—which have been said to mean different things to different people. Further discussion brought forth the explanation that nonparticipating physicians could not be bound by the review procedure and resulting final settlement. The thought was also expressed that use of the words "billed fee" would commit Blue Shield to the point where it would have no recourse.

Dr. Salley then moved to amend the Commission's

recommendation by changing modification "1" to read "Physicians' fees will ordinarily be paid as billed." Modification "2" would be amended to read "Fees which seem to be unusual will first . . .". The word "only" would be deleted from the second sentence of modification "2" since it is superfluous.

Dr. Salley's motion to amend was seconded and adopted.

Dr. Hill's original motion of approval was then adopted as amended.

Dr. Hill stated that the Commission's work had really just begun and that it would soon be considering such matters as plan unification, advisability of separate administrations for Blue Cross and Blue Shield, length of terms for Blue Shield Board members, etc. A request was made that the Commission study the District of Columbia Blue Shield Plan along with others affecting Virginia physicians.

Student AMA

Dr. Murrell stated that he had recently enjoyed a most interesting talk with the President of the Medical College of Virginia Chapter of the Student AMA. The students are most anxious to establish liaison with The Medical Society of Virginia, and have asked Society assistance in setting up programs, permission to print SAMA news in the Virginia Medical Monthly, assistance in setting up "GP Clubs", etc.

The MCV Chapter wants very much to take as many of its members as possible to the annual meeting of SAMA and has requested financial assistance in the amount of $500.

Mention was made of the fact that The Medical Society of Virginia has been sponsoring Senior Day Programs at both medical schools for a number of years. These Programs have been under the direction of the Public Relations Committee and have been well received.

It was then moved by Dr. Weyl that the SAMA request as presented by Dr. Murrell be approved and that the annual Senior Day Programs be continued. The motion was seconded and adopted.

The question was raised concerning the percentage of Virginia students in our medical schools and the figure was placed at 65%. Some states were reported to have higher percentages and the hope was expressed that the Virginia figure could be raised.

Physician Volunteers

Dr. Murrell reported on a meeting with Dr. Robert Duley and Dr. Joseph Rhode, Richmond, with reference to a plan to provide volunteer physician services for physician depleted areas of the State. It was pointed out that there are a number of rural communities which are badly in need of physicians and quite remote from medical facilities. Many of these communities are, unfortunately, not attractive to physicians for a number of reasons.

The objectives of the volunteer program would be:

A. To provide for the medical needs of such communities until full-time physicians can be obtained;

B. Periodically relieve overworked physicians;

C. Encourage physicians to locate in such areas with the knowledge that periodic relief would be forthcoming;

D. Insure full utilization of such facilities that might exist;

E. Revitalize the physician's concept of community service; and

F. Encourage better public relations for medicine as a whole.

Dr. Duley and Dr. Rhode, in cooperation with Dr. Charles A. Pollock, Jr., had drawn a detailed plan of implementation with certain suggested target areas.

Everyone agreed that the proposed volunteer physician program is a very refreshing approach to a long time problem. The feeling was expressed that the physicians concerned should be encouraged to procede. *A motion was then offered by Dr. Grossman which would approve the volunteer physician program in principle and have appointed a committee to further explore the possibilities of such a program and report its findings and recommendations at a future meeting of Council. The motion was seconded and adopted.*

Insurance Forms

Dr. Stone reported that Roanoke physicians have become quite concerned over the great amount of time spent in completing various and sundry insurance claim forms and reports. Dr. Michael Moore of Roanoke has done some rather extensive research on the matter and has stated that the so-called "standardized" forms are actually longer today than they were twelve years ago. He has developed a plan

which would make it possible for all physicians in a particular Society to use a much shorter form. It would require the endorsement of local societies and the cooperation of all physicians.

Council was advised that a proposed form has been developed by the Roanoke Academy of Medicine and that Dr. Stone will make copies available for study. It was suggested that the matter be referred to the Committee on Medical Service for detailed consideration. Dr. Stone stated that the Roanoke Academy would cooperate in every possible manner.

Annual Meeting Format

It was recalled that the 1967 Annual Meeting was something of an experiment inasmuch as a Thursday-Sunday session was tried for the first time. Also, the various specialty groups assisted in arranging the scientific program and two scientific sessions were held simultaneously. It would be Council's decision as to what format would be used for future meetings.

Council was advised that the Program Committee had recently met and made two recommendations. The first was that future Annual Meetings be held from Thursday through Sunday. The majority of members contacted on this suggestion had expressed a strong preference for an end-of-the-week meeting. The Committee's second recommendation was that the format of all scientific sessions be left to the judgment of the Program Committee. Needs and facilities vary from year to year and the Program Committee could most likely do a much better job if its hands were not tied.

A motion to approve the recommendations of the Program Committee was then seconded and adopted.

The Executive Secretary was requested to investigate the possibility of changing those Annual Meeting dates already set in such manner as to comply with the new policy.

"Horizons Unlimited"

The Medical Society of Virginia has been requested to purchase a print of a new film entitled "Horizons Unlimited". This film, prepared by the American Medical Association, would be used in the Health Careers Program of the Virginia Council on Health and Medical Care. The cost would be $100.

A motion to authorize purchase of the film was seconded and adopted.

1972 Annual Meeting

The 1966 Annual Meeting was reported to have been the most popular in the Society's long history. In fact, many members have suggested that consideration be given to meeting in Williamsburg at least every other year. The earliest that the Society could return would be 1972 and Colonial Williamsburg has set aside the dates of November 5-8. These are the only dates available before 1973.

The though was expressed that while more frequent meetings in Williamsburg would seem desirable, great care must be taken that it not be overdone. The feeling was also expressed that the frequency of meetings in Williamsburg should be determined by the House of Delegates.

A motion was then made that the Society hold its 1972 Annual Meeting in Williamsburg and that the dates of November 5-8 be confirmed. The motion was seconded and adopted.

Champus

The Department of Defense has announced that when the Society's current Contract with CHAMPUS expires in May, it will not be renewed. This is because the CHAMPUS program is implementing the "usual and customary" fee concept and a negotiated schedule of maximum allowances is no longer applicable. Consequently, the Contracting Agent will be eliminated and only a Fiscal Agent will be retained.

In order to speed the change over to the "usual and customary" approach, it has been suggested that The Medical Society of Virginia propose cancellation of its existing Contract with CHAMPUS. This would seem to be in the best interests of all concerned. Council was assured that the present Review Committee would remain in effect and that CHAMPUS would refer all unusual and problem cases to it for its recommendations.

Dr. Carmines then moved that The Medical Society of Virginia advise CHAMPUS officials of its willingness to cancel its Contract. The motion was seconded and adopted.

Transplants

Dr. Murrell told Council of the press conference on heart transplants held at Society headquarters on January 22. The conference brought together physicians, clergymen and lawyers and received two-page

coverage in a Sunday edition of the Richmond Times-Dispatch. A story concerning the conference was also picked up by the wire services. Many favorable comments have been received in the State office.

Insurance Review

The President discussed certain weaknesses which seem to exist in the procedure currently used in reviewing claims referred to the Society by the insurance industry. He requested all members to give this matter very careful consideration with the hope that ways and means might be found to eliminate these obvious weak spots.

Chiropractic Legislation

Mr. Miller called attention to a bill before the General Assembly which could compel insurance companies to pay for certain services provided by chiropractors.

A motion was then introduced stating that The Medical Society of Virginia is opposed to legislation compelling insurance companies to pay or reimburse chiropractors under any insurance policy. The motion was seconded and adopted.

There being no further business, the meeting was adjourned.

ROBERT I. HOWARD, *Secretary*

APPROVED:

THOMAS W. MURRELL, JR., M.D., *President*

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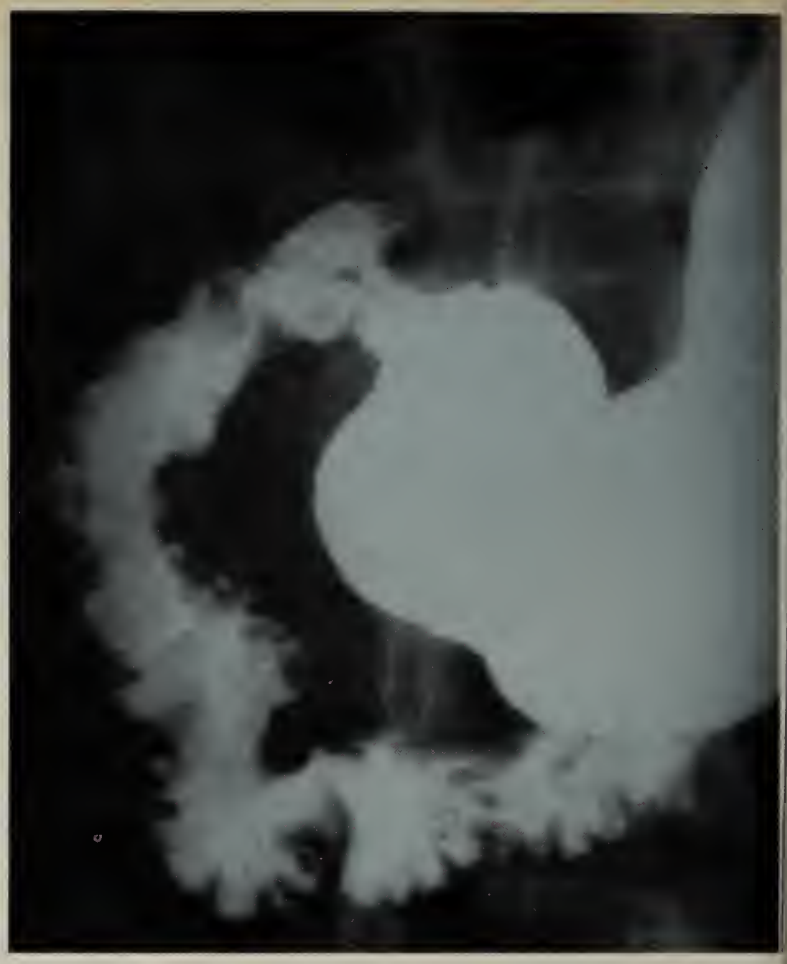
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(1) Bilbao, M. K.; Frische, L. H.; Rösch, J., and Dotter, C. T.: Hypotonic Duodenography, Scientific Exhibit, Radiological Society of North America, Chicago, Nov. 27–Dec. 2, 1966.

(2) Bilbao, M. K.; Frische, L. H.; Dotter, C. T., and Rösch, J.: Hypotonic Duodenography, Radiology *89*:438-443 (Sept.) 1967.

SEARLE

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Woman's Auxiliary....

Reprint from Ohio Medical Auxiliary News

AN AUXILIARY PRESIDENT WRITES TO DR. SLAUGHTER . . . .

Dear Dr. Slaughter:

I am writing in regard to your new book, Doctors' Wives. Before I comment, I want to give you my background so you will see I am not talking without knowledge, as you seem to be. Although I do not write officially, I am sure I voice opinions of most wives of physicians.

My husband is a diplomate of the American Board of Internal Medicine and he and I have been married nearly 36 years. He has read your books (only after they come out in paperbacks) because he has been amazed how your accurate medical knowledge carries over into your novels. Perhaps your knowledge is limited to medicine, however, for it surely does not include medical Auxiliaries.

But back to me: I am a journalism graduate and was engaged in newspaper and public relations work before being married. During World War II I did public relations for the Air Force while Dr. Sauvageot was in service. Since 1946 I have done hospital public relations, primarily for a large city hospital plus special state and national assignments. At present I am Director of Public Affairs for the same hospital so I know doctors well, professionally, as well as socially.

Most important of all, however, is the fact that I have been active in the Medical Auxiliary since I helped organize the Summit County (Ohio) group in 1941. As you can see from the letterhead I am using, I am now president of the Woman's Auxiliary to the Ohio State Medical Association, an office I did not seek but of which I am extremely proud. I have never had time to be a club woman or "joiner" but I feel being an Auxiliary member is not only a privilege but it is a duty—and most enjoyable.

During the past month I have had the distinct pleasure of working in various parts of the country with eight members of the Board of the Woman's Auxiliary to the American Medical Association. Mrs. Karl Ritter, National president, was in the group as was the National president-elect, Mrs. C. C. Long. I led a publications workshop since I am a contributing editor of MD's Wife, the National Auxiliary magazine. It is sent to our nearly 100,000 members. Have you seen the magazine? If not, we might send you a few copies without charge so you can learn more about doctors' wives.

Usually I am brief but I want you to realize I am in an excellent position to know about Auxiliaries and wives of physicians.

I wish you would tell us where any ambitious wife goes around getting votes pledged to back her for the presidency of her State Auxiliary. Because state work is demanding of time, talents and resources, finding qualified women to take responsibility is difficult. Becoming a National officer is not any more political than becoming a president on a state level and the office so many times more demanding. Love of work, sweat (not perspiration), great interest in our husband's profession and the ability to cooperate with one another combine to make Auxiliary leaders on all levels. Your book notwithstanding, our morals are good, our marriage status honorable and we think our guys do all right by us.

If you will look at the education and accomplishments of National Auxiliary presidents and other leaders over the years you will see how your lack of knowledge shows through pitifully in Doctors' Wives.

As Auxiliary members we try to present

a true image of ourselves as women dedicated to service in our communities and to our husbands' profession. Maybe, though, it would be easier to follow the pattern of your questionable characters, most of whom don't seem at all the type of women doctors I know marry.

But maybe the doctors you know are different. If so, I'm glad they are your friends—not mine.

Sincerely,

Ludel B. Sauvageot
(Mrs. J. Paul Sauvageot)
President, Ohio Medical Auxiliary

. . . . AND THIS WAS HIS ANSWER

Dear Mrs. Sauvageot:

Thank you for your letter of October 31. I respect fully your right not to like my book. In fact I shall be very much disappointed if a lot of people, including a lot of wives of doctors, lawyers, dentists, engineers and other professional groups, don't like it. Doctors' Wives is a novel and the characters are imaginary; nevertheless their counterpart can be found in almost any community of a similar size to the fictional community of Weston. It is a matter of medical record that doctors' wives do suffer from emotional illnesses to a degree that is higher than is true of many other groups and of the population in general. If you will get the issue of the American Journal of Psychiatry from the library, to which I refer in the story, you will see that they often come to seek psychiatric treatment for rather serious conditions. I wrote the story in order to dramatize what I consider a disease of civilization, at least what we call civilization here in America. And the distressingly high incidence of divorce among medical families around forty to forty-five amply documents not only the truth of the book's theme but also the need for the story to be told. The fact that you have not come in contact with people like this does not at all mean that they do not exist. I would not expect you to come in contact with them but if you will inquire around in a community the size of Akron, I am sure you will find people with problems just as serious as those presented in the book. They certainly occur in many cities that I know about and you have only to read the morning newspaper for a few weeks to find situations quite as dramatic as the story appearing in real life.

The character of Amy, to which you take such exception, is in fact quite common in our American culture. If you read the book again, I think you will realize that Amy honestly felt that she was helping her husband in the beginning, by concentrating all of her tremendous energies on reaching a high place in the medical auxiliary. None of which is a reflection on the Auxiliary as it exists; my own wife has been a member for many years, wrote the first history of the activities of the organization in Florida, campaigned for candidates whom we felt were sympathetic to medicine and its future, and otherwise promoted auxiliary activities. Nevertheless there are ambitious women whose ambition becomes pathological under just the circumstances depicted in the book; a woman psychoanalyst, and a doctor's wife, with whom I appeared on a radio program in New York, felt that I had, in fact, made Amy's ambition perhaps less strong than really happens in many cases. What I am trying to make clear to you is that, because a fictional character does something you don't like, is no reason to consider that action a slander against everyone in a similar position. Fiction is fiction and fact is fact; I personally wish they weren't so often identical.

Sincerely,

Frank G. Slaughter, M.D.

EDITOR'S NOTE by Virginia President:

The above letters reprinted with permission of their writers, reflect opposite attitudes toward a novel many of you have read, DOCTORS' WIVES. Where this book depicts Auxiliary members in an

unfavorable fashion indeed, we would do well to remember that *it is fiction*. We can and do live above such negative preconceptions of our roles every day. To become overly defensive in this instance would only add fuel to the fire! We can thank Mrs. Sauvageot for making such an apt rebuttal in our behalf.

Conference on Sex Education. Last November in Roanoke, Mrs. Ralph R. Landes and Mrs. Carl Stark represented our Auxiliary at a conference on "Sex Education—A School Responsibility", sponsored by the Virginia Education Association. The program was directed toward primary and secondary educators of our State and afforded an insight into the need of such education and accepted approaches toward instituting such programs into our school systems.

The overall goal of such instruction is to help students accept and value their total self and, in turn, relate themselves to other human beings in a caring, respectful and responsible way. If this can be achieved, sexual behavior will take care of itself.

In many cases while school systems claim to "educate the whole child", they avoid sex instruction. In keeping with this, another speaker stated, "there is no sexuality that is separate from personality."

Sex education in schools should interpret social limits to youngsters who are faced with restrictive puritanical attitudes on one hand, and over glamorized sexual stimuli on the other. Rules of social behavior were referred to as the "glue" of moral values. Sex education courses can effectively explain the "whys" of social rules.

Keynote speaker was Dr. Eleanore Luckey, Chairman, Department of Child Development and Family Relations, University of Connecticut, and a special consultant to the Children's Bureau of the Department of Health, Education and Welfare. Dr. Luckey covered the principal issues during her excellent talk at the opening session, and had interesting points to add when she spoke again the following day as a parent.

Mr. George Burton of the State Department of Education explained that localities must present their proposed programs and materials for sex education to the State Board of Education for approval, and that quite likely approval will be readily granted. Representatives from Arlington, Lynchburg and Roanoke outlined programs which they are developing or have recently introduced into their schools.

Among other fine speakers presented were the Rev. Mr. Churchill Gibson, Jr., Chaplain, St. Stephen's School, Alexandria; Dr. Esther White, Assistant Professor of Health, University of North Carolina; and Dr. Evalyn S. Gendel, State Department of Health, Topeka, Kansas.

This bevy of outstanding speakers repeatedly referred to the "communication barrier". It was agreed that the adult, both parent and teacher, should clarify their own viewpoint of themselves as a sexual human being. In-training for teachers is necessary. The communication barrier then can be broken down by educated and well adjusted leaders from the schools, homes, church and community. Children need help now, and both they and their parents are receptive to sex education programs.

It was felt that such training should begin in kindergarten and continue through the 12th grade. Information should be taught according to the child's age, ability and interest so that a slow, natural growth and discovery of sexuality in self may develop. Also to be desired is the inclusion of sex education into the total curriculum of the school, every teacher dealing frankly with any kind of sexual problem that presents itself in a class. Present instruction has been termed as "band-aid" procedures. It was felt, however, that achievement of the ideal will require considerable time.

Adequate sex education also involves the home and church. These institutions must define their viewpoints and make clear what value system they wish to enforce.

Dr. William G. Thurman, Chairman of the Department of Pediatrics, discussed the medical problems that good sex education can prevent. These included illegitimacy, the high rate of premature births and their resulting medical problems, abortions, venereal disease, and the psychological impact on misinformed or uninformed young people. Dr. Thurman called for better trained teachers and more informed parents. In areas where sex education has been provided, these five major medical problems are all of less incidence, he stated.

Today in this country, three out of 10 schools have sex education courses in kindergarten through the 12th grade. In Virginia 20 school systems have received approval for instructional material.

This conference was of an exploratory nature, its impact to be determined by future courses of action.

Example of Community and Auxiliary Reaction . . . is noted in Danville. At a recent meeting of the Parents' League, Mrs. Ralph Landes participated in a panel program based on ideas garnered at the Roanoke conference. The following day the Medical Auxiliary had a program on the subject presented by Mrs. Henry Bourne and Dr. Frances (Mrs. John) Stoneburner. This generated a lively discussion which continued right through luncheon and for some time afterward.

Soon after this a local radio station featured a discussion between the superintendent of schools, Mr. O. T. Bonner, and a group of high school students. A few days later another group of concerned people met to explore the possibility of forming a chapter of the Virginia Planned Parenthood Association. The ultimate aim of this organization is to assure that every child born in Virginia is a "wanted" child.

It can be hoped that the growing interest in educating young and old toward greater sexual responsibility will eventually lead to the alleviation of many of today's acute social and personal problems.

"Just You" Can Make Difference. "What Can I Do—Just Me?"—This excuse is sometimes used by persons who don't want to bother to register or to vote. To such persons the following reply is given by Edward F. Smith, field representative of The American Medical Political Action Committee. "What can I do?" First, every doctor and doctor's wife must register to vote—but few realize the power of this vote.

"One vote elected as Presidents of the United States, under our electoral college system: John Quincy Adams . . . Thomas Jefferson . . . and Rutherford B. Hayes . . . and President Andrew Johnson escaped impeachment by one vote. One vote made the states of California, Washington, Oregon, Idaho, and Texas, States of the Union. Military conscription was enacted by one vote in 1941. A single vote made France a republic in 1876, and ended it by one vote in 1940. Two governors of Massachusetts have been chosen by one vote.

"During the fight for independence, a bill was presented to the Continental Congress to abolish the English language and adopt German as the official language of this country. That bill was defeated by one vote."

"An even more far-reaching coincidence might be the meeting held in a Munich beer hall in 1923. A fledgling political party held an organizational session to pick a leader. One of the candidates was of conservative philosophy and the other was a firebrand. The latter won by one vote. The party members called themselves Nazis, and the one vote victor of that election was Adolph Hitler.

"Amazing power, the vote? Here's one more incidence of that power. Most persons probably never heard the name Henry Shoemaker, a simple farm lad who lived in Indiana in 1842. That was an election year and a candidate for the state legislature by the name of Madison Marsh solicited the

vote of Henry Shoemaker, and got his promise of support. Late on the afternoon of election day, Shoemaker remembered his promise to candidate Marsh, saddled his horse and rode into town where he cast his single ballot for Marsh. Candidate Marsh went to the Indiana legislature, elected by the margin of one vote.

"A few months later the Indiana legislature was called upon to fill a vacancy in the Senate from that state. In those days U.S. Senators were not selected by popular vote as today. Edward Hannigan was elected to that Senate seat by one vote. Edward Hannigan was elected by the vote cast by legislator Madison Marsh.

"But there is a different finish to the story. Several years later trouble arose between the United States and Mexico and the Senate caucused to determine if the United States should declare war. A vote was taken but it ended in a tie. Senator Hannigan was not present in the chamber for the vote so someone summoned him from his office and he cast the tie-breaking vote, for war. The ensuing war resulted in the annexation of the Southwest Territory to the United States . . . thanks to Mr. Henry Shoemaker, Indiana farmer."

Nationwide Decline in Births Continues

The number of births in the United States continued to decrease in 1966. In that year, only about 3,629,000 babies were born as compared with 3,760,358 in 1965 and 4,257,850 in 1960. Since 1960 the number of births has declined by almost 15 percent for the country as a whole, with the birth rate dropping to 18.5 per 1,000 total resident population in 1966. The total births for 1967 are estimated at about 3,530,000, the sixth consecutive year of decline. The current downward trend is the result of reductions in birth rates for second and subsequent children; the rate of first births has remained at about the same level since 1959. However, it is expected that by 1970 the rate of first births will be rising and once that has happened, increases in birth rates for the second and subsequent children are likely to follow a year or two later. Thus, the recent downtrend in birth rates will be reversed in the next few years as increasing numbers of postwar children reach marriageable ages and begin their own families.

In 1960 every state had a birth rate of over 21.0 per 1,000 population, but by 1966 only four states and the District of Columbia had birth rates as high or higher.

The decline in the number of births has been considerably more marked among whites than among nonwhites. In 1965 the number of white births was 3,123,860, a decrease of 13.2 percent since 1960. On the other hand, nonwhite births fell by only 3.1 percent during the same period. Every state but Nevada had fewer white births in 1965 than in 1960, but in the District of Columbia and 17 states nonwhite births increased. In fact, for these 18 areas as an aggregate, the rise in nonwhite births exceeded 12 percent. (Statistical Bulletin, Metropolitan Life Insurance Company)

Miscellaneous....

Another Doctor Retires—

On January 1, 1968, Dr. Cora Z. Corpening, who has been in general practice in Virginia Beach for fifty years, retired. The Virginia Beach Medical Society had a banquet in her honor on February 3rd and she was presented an award by Dr. Floyd Dormire, who has been in practice there for thirty-five years. Dr. Corpening still plays golf daily at the age of seventy-five. She gave a farewell speech which the Society felt very indicative of the practical philosophy wich has earmarked her life. The Virginia Beach Medical Society was so impressed with her speech and felt it worthy of publication in the Virginia Medical Monthly. It is being published in full, as follows, and the Monthly salutes Dr. Corpening and wishes her much happiness and joy in her retirement:

I feel very unworthy of the honor that the Virginia Beach Medical Society has bestowed on me this evening. It is a heart warming gesture that I deeply appreciate. I feel that through the years, I've done nothing spectacular. I have trod along the same road you are traveling every day, doing the thing that lies next to you, as best you can, and hoping your patients will be benefited by your judgment.

The medical profession has made mammoth strides in the past several decades, especially since World War I, but the basic principles of the practice of medicine have remained the same. Its dedicated physicians have continued to work just as carefully, conscientiously, and untiringly as ever in alleviating pain, suffering and distress.

Our Professor of Therapeutics told us students that there were no more than 20 valuable drugs in the Pharmacopea, and that we should learn to use them wisely. It would be a Herculean task to count the thousands of drugs in use today, and to mention the miraculous cures these drugs have effected.

I was fortunate in seeing probably the first patient treated with Penicillin in Norfolk. I was called to Lynnhaven to see a boy three years old. The year was 1942. I sensed the child was seriously ill, and since his father was an Army Captain, I called the doctor at Fort Story in consultation. The diagnosis was fulminating cavernous sinus thrombosis. We referred the patient to Dr. White who admitted him to Norfolk General Hospital. The child's life was dispaired of, and the Captain was called home. He had heard of this wonderful new drug they used in the service, and demanded it be used on his son, so Dr. White had Penicillin flown in. The daily pictures of the child's face, which were taken, showed the protruding eye and brow gradually receding, and the abnormally high temperature subsided, and his life, I am sure, was saved by the drug.

Many Norfolk doctors saw this interesting patient and marveled at the miraculous recovery. It was sensational then, but today, each one of you see similar incidents from antibiotics every day, and they have become commonplace.

Gone are the days when the doctor carried chloroform and H M C tablets (so called twilight sleep) in his obstetrical bag, and when he delivered most of his patients in their homes, often with only the husband to assist him by holding the kerosene lamp.

On one of these occasions the doctor was busily engaged delivering a baby—he waited a few minutes for the placenta—and a second baby was born, then in rapid succession a third baby's head appeared. This, was just too much for the surprised father who stalked out of the room with the lamp. The irate doctor yelled "What are you doing there, come back here." The man said, "Doctor, I ain't a comin' back. You know, I think it's the light that attractin' 'em."

It is amazing how few infections developed under these circumstances, due perhaps to a higher degree of immunity then than we have now.

It has been interesting to watch the miracle drugs and the vaccines developed and put to use in lengthening life's span. However, when I visit our nursing homes and see the many helpless senile vegetables sitting and lying around, I wonder what has been accomplished in bringing them to this stage. Certainly, arteriosclerosis deserves much research to be done on its prevention and cure. Old age can be interesting and enjoyable, but senility with its handicaps is baffling.

If the Elizabethan period was the Golden Years of England, I feel I have lived through a like period in America. In addition to the pharmaceuticals already mentioned, many other discoveries and inventions transport us to a new and a different world: the automobile, airplane, radio, television, electronics, cable grams, radar, oceanic telephones, and space crafts are but a few.

During the past ten years, Princess Anne County has had a population explosion, and has become a city with many new modern buildings, commercial, educational, professional and domestic. We have also had an inflow of a large number of fine, well-trained, young doctors. What a wonderful opportunity all of you have working in a young, booming, community with a new, well equipped hospital.

I could wish with nostalgia I could erase the years, and start all over again with all your many superlative advantages, but I am contented. I have had a full, busy and rewarding life. I have loved my work, and my association with the medical profession, and my greatest compensation has been their confidence, and the love and appreciation of grateful patients.

Old Generals just fade away, but old doctors, like gray horses, are turned out to pasture. I shall enjoy my freedom from responsibility and shall keep busy foraging in a pasture that is always green. I hope to enjoy family, friends and hobbies I've never had time for before. I might even take off to the bright blue yonder.

I am reminded at this time of the clinic at Black Mountain, North Carolina. The clinic doctor asked a rusty looking old mountaineer who came in how he felt. The old man hesitated a minute and said, "well it's this a-way doctor, I'm still around here, but I ain't a kickin' up any dust."

Studies on the Kinetics of Cellular Proliferation and Growth Rate in Solid Tumors

The cooperation of physicians is requested in the referral of patients with tumors for a study now in progress by the Medicine Branch of the National Cancer Institute at the Clinical Center, National Institutes of Health, in Bethesda, Maryland. The purpose of this investigation is to gain information on rate of cellular proliferation and growth of tumors in man.

A limited number of patients with untreated lymphosarcoma, or reticulum cell sarcoma with tumor readily accessible to biopsy, are of particular interest. Patients with other tumors, particularly lung, will be considered if they have accessible disease. Following the study, patients will be treated when appropriate. When the study is completed the patient will be returned to the referring physician.

Physicians interested in referring patients for these studies should write or telephone: Vincent T. DeVita, M.D., Clinical Center, Room 12-N-236, National Institutes of Health, Bethesda, Maryland 20014. Telephone: 656-4000, Ext. 61480 (Area Code 301)

Editorial . . .

Herbs—Meek Inheritors of the Earth

"O! mickle is the powerful grace that lies in herbs," (Romeo and Juliet, Act II, Scene 3, Line 15)

FEW THERE ARE, I believe, who are not endowed with some of the genes of the naturalist, the agronomist, the gardener and accordingly are not herb conscious.

Is there a mind so devoid of curiosity and wonderment as not to be aroused by the contemplation of a dozen or more different herbs growing in physical contact with each other, and their roots intermingling within a few square feet of soil which constitutes their common source of sustenance, yet each selecting unto itself and assimilating what it selects in such manner as to impart to each plan peculiarly distinguishing characteristics. This, in the vegetable kingdom, is selectivity and discernment of the highest order.

Throughout the annals of the human race so lowly a thing as herbs has been accorded a place of prominence. Indeed, it was an interest in and a search for a more direct route to the source of herbs and spices (the difference in this instance is little more than a matter of semantics) that led to the discovery of the Western Hemisphere.

By botanical definition an herb is any leafy plant without a woody stem. Strictly speaking, spices are the buds, leaves, seeds, bark, roots and berries of various aromatic pungent plants used as flavoring for food, and all of which grow in tropical or subtropical climates. The term spice, however, may correctly refer to both herbs and spices.

It seems reasonable to assume that early the human animal learned what plants to eat and what not to eat from watching birds and wild animals and following their example, as well as from their own trial and error.

In the Bible there are many references to herbs—"Consider the lilies of the fields." There was a time when cumin, a low plant of the carrot family, mint, dill and many others were used to pay tithes. Relatively recently, upon opening the tomb of an ancient Pharaoh, a papyrus was found mentioning safflower and thus establishing that it was in use 3,500 years ago.

Socrates, it would appear, was familiar with hemlock—in fact, too familiar.

Shakespeare made numerous references to herbs. In his day these members of the vegetable kingdom played an important role upon three major counts: (1) primarily as food or, secondarily, for seasoning or flavoring food, (2) as perfumes, (3) and most important of all perhaps, as a source of medicines for the treatment of many ills to which man particularly, but other animals too, were and are prone. Thus is one key to the classification of herbs provided, i.e., a classification based upon the purpose for which used.

A second classification of herbs takes into account the growth habit of the plant, i.e., whether annual, as anise, basil, dill; biennial, as caraway, celery, parsley; or perennial, as chive, sage, thyme. A third classification is according to family, as mint, aster or lily. A fourth system of differentiation derives from the part of the herb in demand, i.e., whether it is the leaf, the bud or flower, the seed, the root or the essential oil (usually from the seed) that is utilized. The leaves of sage, mint, basil, marjoram and tarragon; the bud and flower of saffron; the seed of anise, fennel and caraway; the roots of ginger, horseradish and ginseng; and the essential oils of anise, safflower and wormwood are examples of this latter categorization of herbs. Still another classification could be established in terms of which can be raised from seeds and which require the transplantation of plants or roots. Tarragon is one herb virtually impossible to start from seed.

The dried leaves of certain herbs, notably those of sweet marjoram and basil have been used for snuff when snuffing was a popular practice among the gentry. Moreover, such herbs as saffron, safflower and common poke (its berries) have been used as sources of vegetable dyes.

The North American Indians played an important role in the development of herbs in this country. They were highly knowledgeable in the use of plants for food and medicine. Various plants native to their regions were used by different tribes depending upon the area in which each tribe lived. Ginseng was a great favorite with the Iriquois; they used it for most ills. Wormwood, from which comes the oil essential in the making of absinthe, was used by the Zunis, a pueblo tribe in western New Mexico. Dandelion and black snake root were popular with the Chippewas, while in the southwest the Patago tribe used macerated mesquite twigs.

It is an historical fact that the colonials who came to America brought with them their herbs and seeds. As early as 1775 basil seed was advertised for sale in the Virginia Gazette. The herb lore of the Indians naturally influenced greatly the Americans' use of certain herbs for medicines. The Indians knew which ones were effective against what. They also knew how to preserve meat with tansy and what plants to gather for dyes.

After the "Boston Tea Party" it is said to have been unpatriotic to drink black tea imported from England. So, the New Englanders tried the Indian drink of red bergamot (an herb of the mint family) called Oswego tea. (Straying briefly from the realm of herbs, I would have suggested spicebush or sassafras tea. I've drunk gallons of both.)

It will, of course, not be forgotten either that it was the Indians who first cultivated tobacco, a plant conforming to the definition of an herb, and introduced it to the White Man. In so doing, he started something, and although undoubtedly unwittingly, he exacted a large measure of revenge for the depredations visited upon his race by the intruders from afar. This revenge continues and there is no prospect of its relentment.

To attempt a review, or even a listing, of the various herbs defined by the U. S. Department of Agriculture would be wholly superfluous

to the purpose of this commentary. There are, for example, 50 or 60 varieties of basil and 30 varieties of marjoram.

For such interest as may still stem from the medicinal use of herbs, may it suffice to say that recently a leading authority on herbs in this country reported that about 80 perennials grown in gardens today are to some extent used in current medical practice. It is not known whether he included the Mexican beet from which is extracted a corticosteroid essentially equivalent to that obtained from the adrenal glands of slaughterhouse animals. It nevertheless is a fact that this beet is a source of difluoro-hydroxyprednisolone-acetonide.

In any event, the urge at least to mention some of the more common herbs indigenous to the Ragged Mountains of Virginia, among which this writer spent his formative years, is irresistible.

Sage (the most maligned of all herbs because of an almost invariable practice on the part of cooks to use too much of it), mint, horehound, catnip, wild onions (let the nobility and the bourgeoisie call them chives, if they will, but to the rustic hoi polloi a chive "ain't nothing" but a "bodacious" wild onion), purslane (pursley to the seldom-bathed sons of the soil who, since yesteryear, have gradually disappeared from the American scene), dandelions (he who has never been drunk off of dandelion wine is fortunate—and may he so remain), wild mustard, peppergrass, field cress (sometimes called "creeces"), mullein (commonly pronounced "mullin"), "life-everlasting" or "rabbit tobacco" (Its authentic botanical or generic name, according to the U. S. Department of Agriculture's informational service, is Gnathalrum Obtusifolium, but by any name its sage-like odor was as familiar to many as that of new mown hay and its smokableness was equally as well known to all of my mountaineer associates—both girls and boys. With much relish, I've smoked my share of it.), poke, pennyroyal and lo! the loathsome and ubiquitous chenopodium were all as common as pig tracks in the fields and garden of my boyhood home. Almost certainly chenopodium would be found around the base of any old ancillary structure and the garden fence of most rural homesteads of my acquaintance in Albemarle County, Virginia. Many an ascaris—and hookworm (Necator americanus)—too—has, by reason of a mass of chenopodium (vermifuge) seeds "done up" in sorghum or "blackstrap" molasses to form a ball, having been damned down their gullets, parted company with about all of the "Joes" and "Janes" of my generation in the hill country.

Somewhat less common, or at any rate found only in environments peculiarly favorable to their growth, were such herbs as mayapple, the source of podophyllum, jack-in-the-pulpit, calamus, snakeroot—with the odor of a snake—polecat cabbage that, as the name implies, simply stunk, foxglove, whose dried leaves constitute the source of digitalis, sarsaparilla (listed as a tropical American smilax, but it nevertheless grows in temperate zone greenhouses or cold frames), from the roots of which a pleasing flavor comes, nightshade (deadly nightshade), from whose roots and leaves comes belladonna with its active principle, atropine. This poisonous herb was not uncommon; nor was hemlock, a

garden herb of the carrot family, in the yards, gardens or around woodpiles of old homes in Virginia.

Pipsissewa, of the genus Chimaphila, the active principle of which is chimaphillin, once vaunted as a cancer remedy and as a diuretic, was one of the first herbs with which I became acquainted in the woods near our home; and this was because our colored woman, who could neither read nor write, knew this plant, called it by its authentic name and knew its properties. She used to "flush her kidneys," and those of anyone around interested in having their kidneys flushed, through the efficacy of her pipsissewa tea.

Lastly, I came to the king of the herbs that grew in the mountain coves within my boyhood range of exploration. This was ginseng, previously mentioned as a favorite herb of the Iriquois Indians. Now! to me, here was and is the paragon of herbs. Ginseng (from the Chinese Jin Tsan, life of man) was thought by the Chinese to be a potent remedy for practically all ills, but particularly was it valued as a restorative and as an aphrodisiac. It is grown in China but that grown in America was considered much superior to the Asiastic variety. The roots of ginseng are extraordinarily irregular and often of bizarre shape. The more nearly the shape of the root—or one of the several roots of a single plant—resembled that of the human body (a not unusual finding, if one's imagination was sufficiently elastic) the more highly was it valued by the Chinese.

During my youth ginseng grew in the shaded coves, nooks and crannies of the Blue Ridge and Ragged Mountains. As a means of earning a livelihood, it was hunted by a few individuals, with all of whom disreputableness was a common hallmark. Various and sundry acts of skulduggery within their scrounging area were quite likely to be attributed to ginseng hunters. As the herb became increasingly more rare in the wild state, but its demand no less lively by the Chinese, it was, to an appreciable extent, for some time raised under a lattice screen to protect it from the direct rays of the sun. As late as the third decade of this century there was operated in Crozet, Virginia, by a citizen of that village, a sizable ginseng arboretum. Although a demand for it—almost entirely by the Chinese—remained great and the price high, its cultivation, due largely no doubt, to the cessation of trade directly between the United States and China, has markedly declined. However, an occasional patch of ginseng may still be found in the mountain fastnesses once upon a time familiar to me.

When, after imponderable millennia, the world over all of its surface, except for a belt at the equator, will have grown too cold to support either vegetable or animal life, the two bugs someone has envisaged, preening their antennae in the cherry-red glow of the noonday sun, may be perched upon an herb. That herb, in all probability, would be a low plant with three leaves at its top—each leaf being made up of five leaflets —with either small greenish-yellow blooms or scarlet berries (depending upon the season). Thus would it meet the characteristics of ginseng. For it, ecologically, the situation would be ideal.

H. Lamont Pugh, M.D.

The Guest Editorial

FROM TIME TO TIME readers of the Virginia Medical Monthly mistake the role of the guest editorial which appears in each issue of the journal. The most recent misunderstanding of this sort occurred following the guest editorial by Dr. R. F. Archambault of Wayne, Michigan, which appeared in February. This dealt with various aspects of hospital administration and costs. (See Letters to the Editor.)

In order to clarify this it should be pointed out that two kinds of editorials appear each month. The *guest* editorial is found near the front of the journal between the advertisements and the scientific articles. These are written by physicians, nurses, dentists, pharmacists, hospital administrators and laymen who have some connection with medicine. Several years ago a guest editorial by a mortician proved helpful. These short articles have been a popular feature for many years. They usually arrive unsolicitated, but at times it becomes necessary to solicitate guest editorials when the supply falls dangerously low.

These editorials represent the opinion of the writer and do not necessarily represent the policy of the Virginia Medical Monthly or The Medical Society of Virginia for which the journal serves as its official organ. The journal welcomes different views on subjects of major interest and at times this means a guest editorial may be at complete variance with the policy of the Society and the general feeling of the membership. Several years ago an article was published that had kind words to say about socialized medicine. We trust few readers were misled to believe that The Medical Society of Virginia had decided to embrace socialized medicine. On the other hand a guest editorial may appear so worthwhile that an accompanying regular editorial may appear in the same issue of the journal. An example of this occurred several months ago about one aspect of nursing.

The *regular* editorials which are located between the scientific articles and the advertisements in the back of the journal fall into a different category. These are written by members of the Editorial Board and in general they reflect the policies of The Medical Society of Virginia. In view of the variety of topics dealt with in editorials, and the limited number of subjects discussed by the Council and the House of Delegates, it is more factual to say that they endeavor not to run counter to the known policies of the Society. This is not difficult, as a rule, for while physicians are notoriously individualistic, the practitioners of Virginia are a fairly homogeneous lot and think more or less alike about the major problems that beset medicine. It may be mentioned in passing that the Society has no policy regarding herbs. (See preceding editorial.)

It is hoped that this explanation will clarify the difference between the guest and regular editorials. It is also hoped that more of the readers of the Virginia Medical Monthly will feel that they have a message to convey to the rest of us and will send in guest editorials to the journal. There are a few subjects though, we wish our contributors would avoid. Please use discretion in your choice of topics—for just now we have an adequate backlog of headaches as it is.

H.J.W.

News....

Calendar of Events

Cardiovascular Responses to Anesthesia—Fifth Annual Spring Symposium of Virginia Society of Anesthesiologists—Sheraton Motor Inn—Richmond—April 19-21, 1968.

Virginia Society of Ophthalmology and Otolaryngology—49th Annual Meeting—The Cavalier—Virginia Beach—May 2-4, 1968.

The Child With Heart Disease—Recognition and Management—Continuing Education Program of the Medical College of Virginia—Richmond—May 9-10, 1968.

Virginia Academy of General Practice—Annual Scientific Assembly—The Chamberlin—Fort Monroe—May 9-12, 1968.

Medical College of Virginia Alumni Scientific Assembly—Richmond—May 31, 1968.

American Medical Association—Annual Meeting—San Francisco, California—June 16-20, 1968.

Seaboard Medical Association—Annual Meeting—Nags Head, North Carolina—June 21-23, 1968.

Medical Aspects of Sports—Sponsored jointly by The Medical Society of Virginia and Virginia High School League—University Hall—Charlottesville—July 21, 1968.

9th Annual Cardiovascular Symposium—Sponsored by Tidewater Heart Association and Council on Clinical Cardiology, American Heart Association—Lake Wright Motel—Virginia Beach—September 6-7, 1968.

New Members.

The following new members were received into The Medical Society of Virginia during the month of February:

Ralph Harrison Boulware, M.D., Lynchburg
Robert Henry Bowden, Jr., M.D., Lynchburg
Francisco Cardenas, M.D., Arlington
Beverly Noe Chambers, M.D., Berryville
Elia Charles Dimitri, M.D., Charlottesville
Milton Harris Donaldson, M.D., Charlottesville
John Thruston Farrar, M.D., Richmond
William Paul Grigsby, M.D., Weber City
Hobert McKinley Hampton, Weber City
Luis G. Maldonado, M.D., Clifton Forge
Carolyn Jean Martin, M.D., Richmond
Meyer Steingold, M.D., Norfolk
David Deaderick Stone, M.D., Charlottesville
Melvin Earl Yeamans, M.D., Richmond

Fairfax County Medical Society.

Dr. C. Barrie Cook, Fairfax, is president of this Society, having been installed January 1st. President-elect is Dr. Thomas M. Wright, Falls Church; vice-president, Dr. Donald S. Thorn, Annandale; secretary, Dr. Rolf A. Koehler, Fairfax; and treasurer, Dr. R. Alan Mackintosh, Vienna. Dr. Edward J. Gallagher Fairfax, Dr. William L. Bekenstein, Fairfax, and Dr. Alberto J. Garcia, Falls Church, are members at large of the executive committee.

Northampton County Medical Society.

Dr. William F. Bernard has been elected president of this Society and Dr. W. J. Sturgis, Jr., secretary. Both are of Nassawadox.

Physician Saves Family in Fire.

Dr. Ramon Garcia, Falls Church, was awakened recently by the coughing of his six year old daughter and discovered the house was on fire. He took his wife and daughter outside then went back for the two sons. Dr. Garcia said he stayed close to the floor and held his breath as long as possible to avoid being overcome by smoke. He half-carried and half-dragged the boys from their beds to the street. The fire apparently started in the recreation room or garage and did about $50,000 in damage.

The Virginia Diabetes Association

Will hold a program meeting in conjunction with the Virginia Academy of General Practice at the Chamberlin Hotel, Fort Monroe, May 9-12. The following program will be presented: Hypoglycemia—Prevention and Treatment by Dr. John A. Owens, Jr., University of Virginia; Outpatient Management of Impending Diabetic Acidosis by Dr. James M. Moss, Alexandria; Diabetes and Surgery by Dr. John A. Galloway, Medical Research Division, Eli Lilly and Company, Indianapolis; and Management of Insulin Allergy and Resistance by Dr. Buris R. Boshell, Medical College of Alabama, Birmingham.

Dr. H. St. George Tucker, Richmond, is president of this Association, and Dr. Bernard H. Miller, Norfolk, secretary-treasurer.

Symposium on Diabetes Mellitus.

The Georgetown University Hospital, Washington, D. C., will hold a symposium on Current Concepts in the Etiology and Treatment of Diabetes Mellitus on May 24th. The program is: Mechanism of Action of Insulin by Dr. Rachmiel Levine, New York Medical College, with Dr. Melvin Blechner as discussant; Diabetes and Exercise by Dr. Maurice S. Goldstein, Michael Reese Hospital, Chicago, and discussant Dr. Dewitt E. DeLawter; Mechanism of Insulin Secretion by Dr. David M. Kipnis, Washington University, St. Louis, with Dr. Frederick Wolff as discussant; Glucose and Plasma Insulin in Early Diabetes by Dr. Stegan S. Fajans, University of Michigan, Ann Arbor, with Dr. Lillian Recant as discussant; Microvascular Disease in Diabetes by Dr. Marvin O. Siperstein, University of Texas, Dallas, with Dr. James M. Moss as discussant; and Retinopathy and Neuropathy in Diabetes Mellitus by Dr. Richard A. Field, Jefferson Medical College, Philadelphia, with Dr. John J. Canary as discussant. All discussants are members of the faculty of Georgetown University.

Hospital Staff Members.

Dr. R. N. Shelley has been elected chief of the staff and chief of surgery at the Norton Community Hospital in Norton. Dr. U. S. Gonzalez is alternate chief of staff and chief of medicine; Dr. F. S. Booth, chief of obstetrics; Dr. D. S. Park, chief of pediatrics. Dr. D. B. Jones, secretary of the medical staff, and Dr. Joseph Straughan program secretary.

Dr. Otis W. Doss has been elected president of the staff and chairman of the executive committee of the Northampton-Accomack Memorial Hospital, Nassawadox, Dr. R. R. Novoa is vice-president and Dr. F. L. Beckel, secretary.

Fellows of American College of Radiology.

The following Virginians received the Fellowship Degree of the American College of Radiology at its meeting in Chicago in February:

Dr. Powell G. Dillard, Lynchburg.

Dr. William F. Weller, Roanoke.

Dr. Noland M. Canter, Jr., Harrisonburg.

Forum for Child Psychiatry.

The Sixth Annual Spring Forum for Child. Psychiatry will be held in Baruch Auditorium of the Medical College of Virginia on May 31st. This is sponsored by the Division of Child Psychiatry of the College

and the Virginia Treatment Center for Children. It is being held in conjunction with the meeting of the Southeastern Region of the American Association of Psychiatric Clinics for Children.

A Post Graduate Cardiology Conference

On "The Child With Heart Disease: Recognition And Management" will be presented at the Medical College of Virginia, Richmond, on May 9-10.

This course is designed for the pediatrician and practitioner of medicine who may encounter heart disease in children. It will illustrate modern methods of diagnosis and management in children with cardiac problems. Lectures by outstanding guest speakers will be supplemented with television presentations and open discussion.

Pediatricians, radiologists, physiologists and surgeons will participate in small group conferences with patients. Emphasis will be placed on clinical tools and modern laboratory methods of diagnosis.

Illustrative cases will be selected from cyanotic newborns, infants with heart failure, children with congenital lesions (especially those amenable to surgery), rheumatic fever and asymptomatic children with possible cardiac disease.

The Virginia Dermatological Society.

At the annual meeting of this Society, held in Charlottesville, November 19th, Dr. Carl Lingamfelter, Richmond, was elected president, Dr. Harry Pariser, Norfolk, vice-president, and Dr. Peyton Weary, Charlottesville, secretary-treasurer.

The next annual meeting will be held in conjunction with the annual meeting of The Medical Society of Virginia in Roanoke, October 13th. All interested physicians are invited to attend the scientific portion of the meeting which will begin at 9:00 A.M.

Virginia Association of Medical Assistants

The Executive Board of The Virginia Association of Medical Assistants met January 21, 1968 at the Hotel Roanoke. It was the first meeting of the Board under the new president, Mrs. Doris Jennings, who conducted the meeting in an efficient and business-like manner, despite several perplexing problems and situations.

Mrs. Martha Welch passed the Certificacation Examination in 1967 and is now a Certified Medical Assistant. This is quite an accomplishment because there are only two in Virginia. Interested persons may obtain Certification Packets from each chapter president. The testing center at present is R.P.I. However, the Certifying Board will arrange for the examination to be conducted in almost any geographical location provided there are a sufficient number of applicants in that area. Last year, 74 members obtained their C.M.A. Certification. Come on Doctors, let's urge and assist our ladies to become Certified Medical Assistants in 1969!

Doctors, please mark these dates on your calendar for coming events of interest to those ladies in your offices who are members of their local, state and national organization. Please allow them time off to attend these meetings and also try to encourage more members of your Staff to join.

1. Virginia Academy of General Practice—May 9-12, 1968, Hotel Chamberlin, Fort Monroe.
2. Virginia Association of Medical Assistants Educational Seminar, May 18-19, 1968—Portsmouth.
3. Virginia Association of Medical Assistants Board Meeting, August 18, 1967—Roanoke.
4. The Medical Society of Virginia, October 13-16, Hotel Roanoke.
5. Virginia Association of Medical Assistants Convention, November 8, 9, 10, Cavalier Hotel, Virginia Beach.
6. American Association of Medical Assistants—October—Columbus, Ohio.

Mrs. Jeanne Mitchell, a very capable and hard working Public Relations Chairman, and her Committee are already working on

plans to assist with the annual meeting of the Virginia Academy of General Practice and The Medical Society of Virginia in addition to operating a Booth at each of these meetings.

JOHN WYATT DAVIS, JR., M.D.

Home-Office for Sale.

In exclusive Chevy Chase, Washington, D. C. One block off Connecticut Avenue at Chevy Chase Circle. Six room office suite, four bedrooms, formal living and dining rooms, etc. Call owner (202) 244-3327. (*Adv.*)

Medical Office Space

To rent or share. Attractive, clean offices in Sterling Park and Herndon available. Call Mr. Foley (703) 471-4491 for information. (*Adv.*)

Virginia Beach

Space available in attractive medical office building of colonial design, currently under construction in one of the fastest growing subdivisions in Virginia—Windsor Woods. Call or write Wilson Realty Company, Post Office Box 1016, Virginia Beach, Virginia 23451. Phone 425-1000, nights, 428-7853. (*Adv.*)

Hospital Physicians Wanted.

The Medical Staff, Board of Directors, and Administration of Winchester Memorial Hospital, Winchester, Virginia, are agreed to a plan of four full-time physicians to be in private general practice limited strictly to the Emergency Department of the Hospital. Average work week is forty-two hours. A generous guarantee has been established.

Four experienced physicians having a Virginia license are needed. The target date is July 1, 1968. Interested physicians contact the Committee Chairman, James A. Miller, M.D., Box 554, Winchester, Virginia. Telephone 662-0377 (area code 703). (*Adv.*)

Wanted.

Qualified anesthesiologist to head department at C & O Hospital, Clifton Forge, Virginia. Income in excess of $30,000, largely on fee for service basis. Apply to J. M. Emmett, M.D. (*Adv.*)

Location Wanted.

Anesthesiologist, FACA, wanted relocation to State of Virginia. Tidewater area preferred. Licensed, experienced, fee for service preferred, group or partnership accepted. Write #135, care Virginia Medical Monthly, 4205 Dover Road, Richmond, Virginia 23221. (*Adv.*)

Obese Children,

Ages 8-14, will be accepted in a summer camp sponsored by the Department of Pediatrics at the University of Virginia. We have places for 40 boys and girls. Tuition $200.00. Camperships available. Write to Peter Houck, M.D., Box 83, University of Virginia Medical Center, Charlottesville, Virginia 22901. (*Adv.*)

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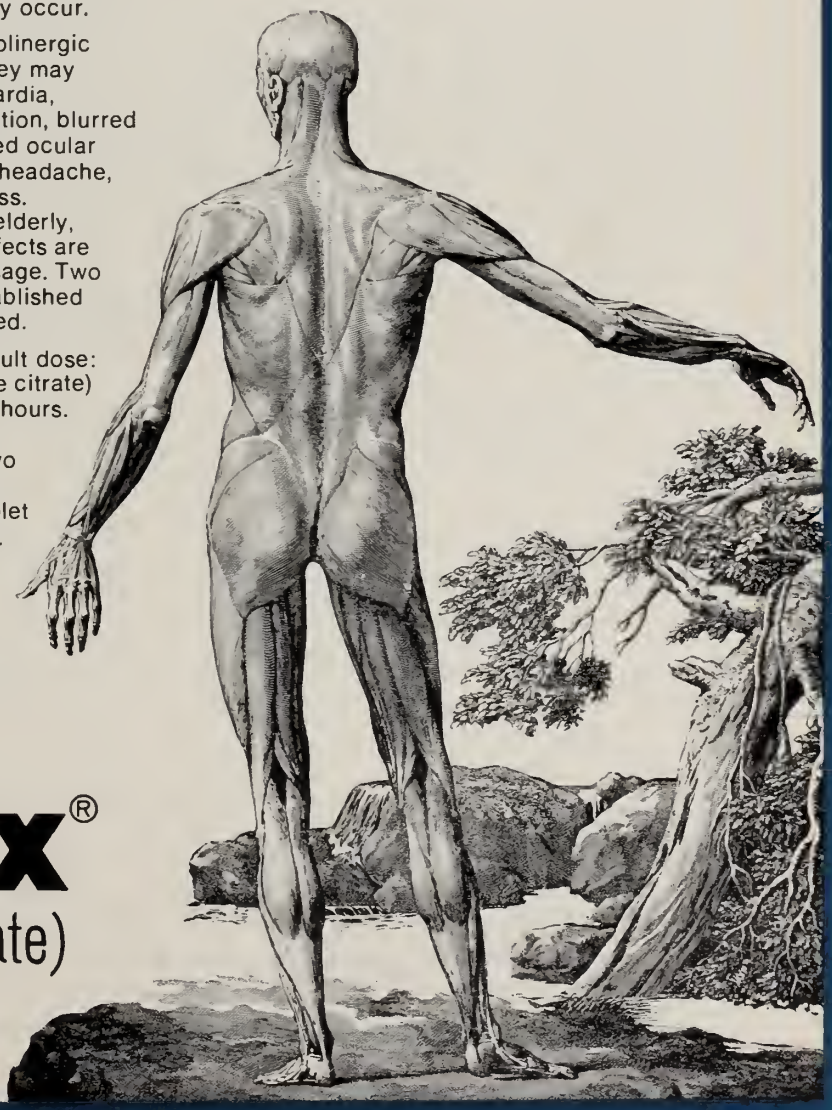
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Obituary....

In Memory of William Eugene Lynn, M.D.

Dr. William E. Lynn was born in Brunswick, Maryland, March 16, 1914. He was a graduate of Brunswick High School, the University of Virginia pre-medical and medical schools from which he received the degree of Doctor of Medicine in 1936. He served his internship at the United States Marine Hospital, Baltimore, Maryland, the University of Maryland Hospital, and Sydenham Hospital, the latter a contagious disease hospital affiliated with Johns Hopkins.

Dr. Lynn was truly a beloved physician. He was never known to raise his voice to a patient, never known to lose patience, never known to refuse to see a patient in his office, in the patient's home, or in the hospital. He treated the wealthy, the middle class, and the poorest of the poor regardless of race. creed or color with absolutely the same kindness, patience, and forbearance. He always gave every patient the very best of which he was capable. Whenever there was a slightest doubt as to his diagnosis or therapy he invariably consulted freely and with the best people available in order to give the patient the utmost in care and consideration. He was truly a dedicated, beloved physician and friend.

On the night of the 31st of October, 1967, Dr. Lynn was driving into the driveway of his country home. An unknown enemy had placed two baskets in the driveway. Mrs. Lynn got out of the car to remove the baskets so that Dr. Lynn could drive on into his home when the assailant, camouflaged completely in a thick hedge immediately adjacent to the driveway, shot into the car and Dr. Lynn was killed immediately. The assailant made a quick retreat and has not been found at least until the day of this dedication of the Lynn Care Center on the 25th of February, 1968.

Dr. Lynn's death represents a tragic loss to his family, to his friends, to his professional associates, to his patients, and to Front Royal and Warren County in general. His constant readiness to extend a kind word or a hand of greeting to every person he encountered will be sorely missed for years to come.

A resolution has been adopted by the Board of Trustees and the Medical Staff of the Warren Memorial Hospital and it has been decided to name the extended care center adjacent to the Warren Memorial Hospital in honor of our friend and co-worker. The resolution passed reads as follows:

WHEREAS, Dr. William Eugene Lynn has served the citizens of Warren County as physician for more than thirty years, and;

WHEREAS, his capable devotion to his patients and the medical profession has distinguished him as a gentleman, physician and friend, and;

WHEREAS, his contributions to the community have earned him a place of prominence, admiration and respect, and;

WHEREAS, it is the desire of the Board of Trustees and the Medical Staff of Warren Memorial Hospital to pay a special tribute to Dr. Lynn; therefore be it

RESOLVED, that the Warren Memorial Hospital Annex be named the LYNN CARE CENTER; and be it

RESOLVED, that a bronze plaque of this resolution be prepared and placed in the annex, and be it further

RESOLVED, that a copy of this resolution be prepared and presented to Mrs. Lynn.

A copy of this resume is to be placed in the cornerstone of the Lynn Care Center, a copy is to be presented to Mrs. Lynn; one is to be included in the minutes of the Medical Staff of the Warren Memorial Hospital, another to be placed within the minutes of the Medical Society of Northern Virginia, another to be sent to the Virginia Medical Monthly, and a record of this death is to be inserted in the Journal of the American Medical Association.

Dr. Alexander.

The year 1967 brought to Southside Virginia the death of an eminent physician, Dr. Haddon C. Alexander, Jr., specialist in diseases of the eyes, ears, nose, and throat. It is with sadness we review his death and with distress we note his departure from our medical staff.

Born August 7, 1897, at Old Church, Hanover County, he attended the public schools of that county and then enrolled in Custer Springs Academy near South Boston. He attended Hampden-Sydney College for three years, took supplementary courses at the College of William and Mary, and was permitted to enter the Medical College of Virginia without obtaining an undergraduate degree. After an illness, he graduated from the Medical College of Virginia in 1926, completing his medical internship there in the same year. He served one year of residency, 1926-27, at the Episcopal Eye, Ear, Nose and Throat Hospital in Washington, D. C., and a second year, 1927-28, at the Medical College of Virginia in the ear, nose and throat department. In 1928, Dr. Alexander opened his office for practice

in Farmville but stayed only one year before moving to Newport News. In the spring of 1930, he was challenged by the lack of coverage for his specialty in the Farmville area; and with personal confidence that the area needed him, he returned to Farmville. He practiced here until his retirement on August 1, 1966, and died on November 4, 1967, at the age of 70.

Dr. Alexander was proud of the community in which he had placed great confidence and exhibited this through his civic work with the Farmville Area Development Corporation, the Farmville Recreation Association, the Lions Club, the Boy Scouts, and other athletic groups for youth. He availed himself of professional contacts and was a member of the American Medical Association, the Southern Medical Association, The Medical Society of Virginia, and the Fourth District Medical Society.

A man known and respected by many people in all walks of life, Dr. Alexander was a dedicated, skilled, and conscientious physician whose retirement was sorely felt in the loss of his service to us, and whose death is a reminder of the mortality of us all. His legacy to us is a son dedicated to the service of mankind through medicine, and hundreds of Southside Virginians who both see and hear better because of his efforts.

Be It Resolved: that the Southside Community Hospital Medical Staff enter into its minutes these remembrances of Dr. Alexander and that a copy be sent to The Medical Society of Virginia, and to his wife and son.

H. B. Holsinger, M.D.
Ray A. Moore, Jr., M.D.

Dr. Siersema.

Dr. Reynold Clinton Siersema, Richmond, died on November 27, 1967. He is survived by his wife, one son and two sisters.

Dr. Siersema was born August 24, 1900, in New York City. He was a graduate of Panzer College and Juaniata College, where he received the B.S. Degree; and the Medical College of Virginia, where he was awarded the M.D. Degree in 1934.

Dr. Siersema began his career as a Professor of Physical Education at the College of William and Mary. In 1927, he became coach of athletics and head of physical education at Juaniata College, where he remained until 1930. After graduating from medical school, Dr. Siersema served his residency in general surgery in Grace Hospital, Richmond, and had continued to practice there since that time. He served as Assistant Professor of Clinical Surgery at the Medical College of Virginia, as well as visiting surgeon at McGuire Veteran's Hospital. His fraternal memberships included Lambda Chi, social fraternity, Omicron Delta Kappa, honor fraternity, and Phi Epsilon Kappa, Sigma Delta Psi, and Phi Chi, professional fraternities. Dr. Siersema was a Fellow of the American College of Surgeons, a Fellow of the Southeastern Surgical Congress, and a Diplomate of the American Board of Surgery. He was a 32nd Degree Mason, and a Shriner. During World War II, he served as Assistant Chief of Surgery, 8th Evacuation Hospital, with the rank of Lieutenant Colonel, United States Army. He was awarded the Bronze Star for his services. He was a communicant of St. Stephen's Episcopal Church in Richmond. He continued to use his physical education training in his recreational pursuits and was devoted to all sports.

During his medical school days, Dr. Siersema was player-coach of the basketball and baseball teams at Medical College of Virginia. At that time, his ambition was to complete medical school and an internship, and then return to William and Mary as Medical Director of sports and physical education. However, he became interested in surgery and decided to pursue this specialty.

Dr. Siersema will be remembered for his sympathetic and understanding manner with his patients, nurses, and fellow physicians. His life was totally dedicated to the practice of his profession, and he will be long remembered by his associates and patients.

Therefore Be It Resolved That this brief record of the life of our friend and associate be spread on the minutes of the Richmond Academy of Medicine, and that copies be sent to the members of his family, and to the Virginia Medical Monthly.

Wm. R. Hill, M.D.
Robley D. Bates, M.D.
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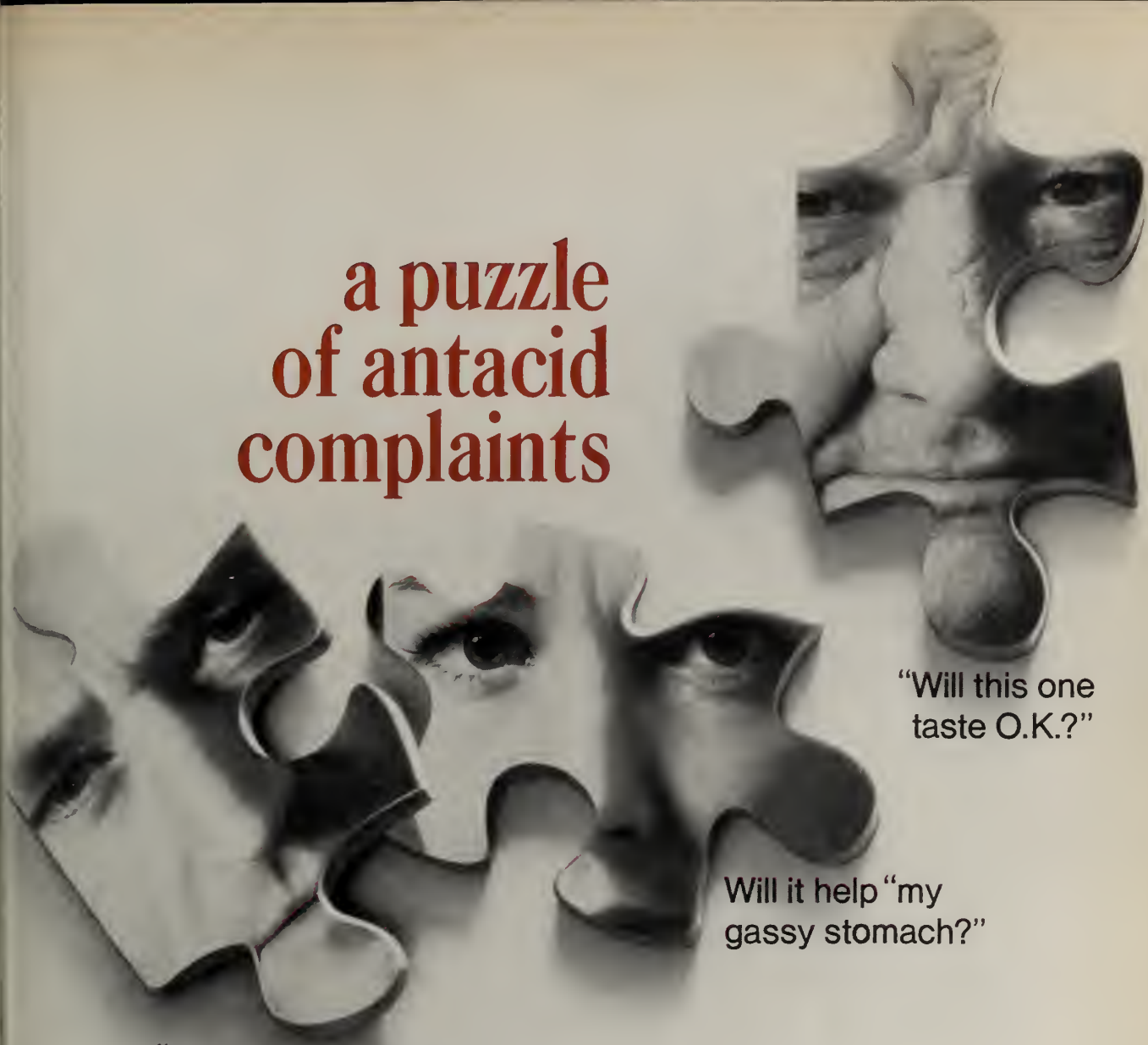
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References: 1. Shumon, C.: Am. J. Med. Sci., 225:54, 1953. 2. Perchuk, E., et al.: Angiology, 12:102, 1961. 3. Rowls, W., et al.: Med. Times, 87:818, 1959.

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References: 1. Hoon, J. R.: Arch. Surg. *93*:467 (Sept.) 1966.
2. Danhof, I. E., Personal communication.

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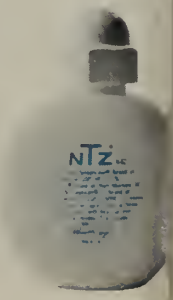
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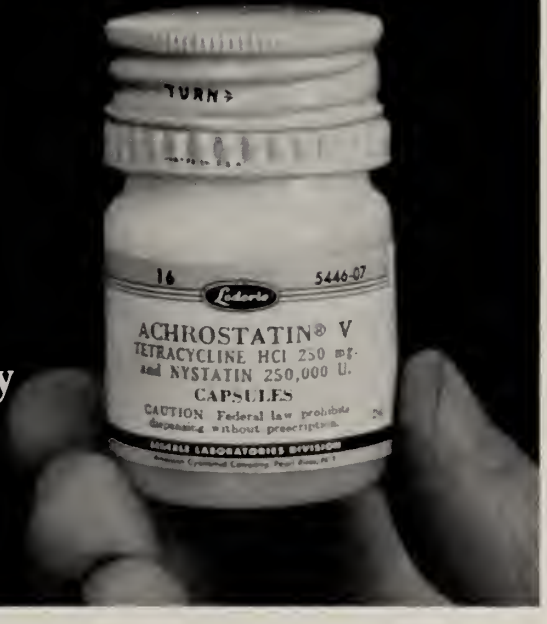
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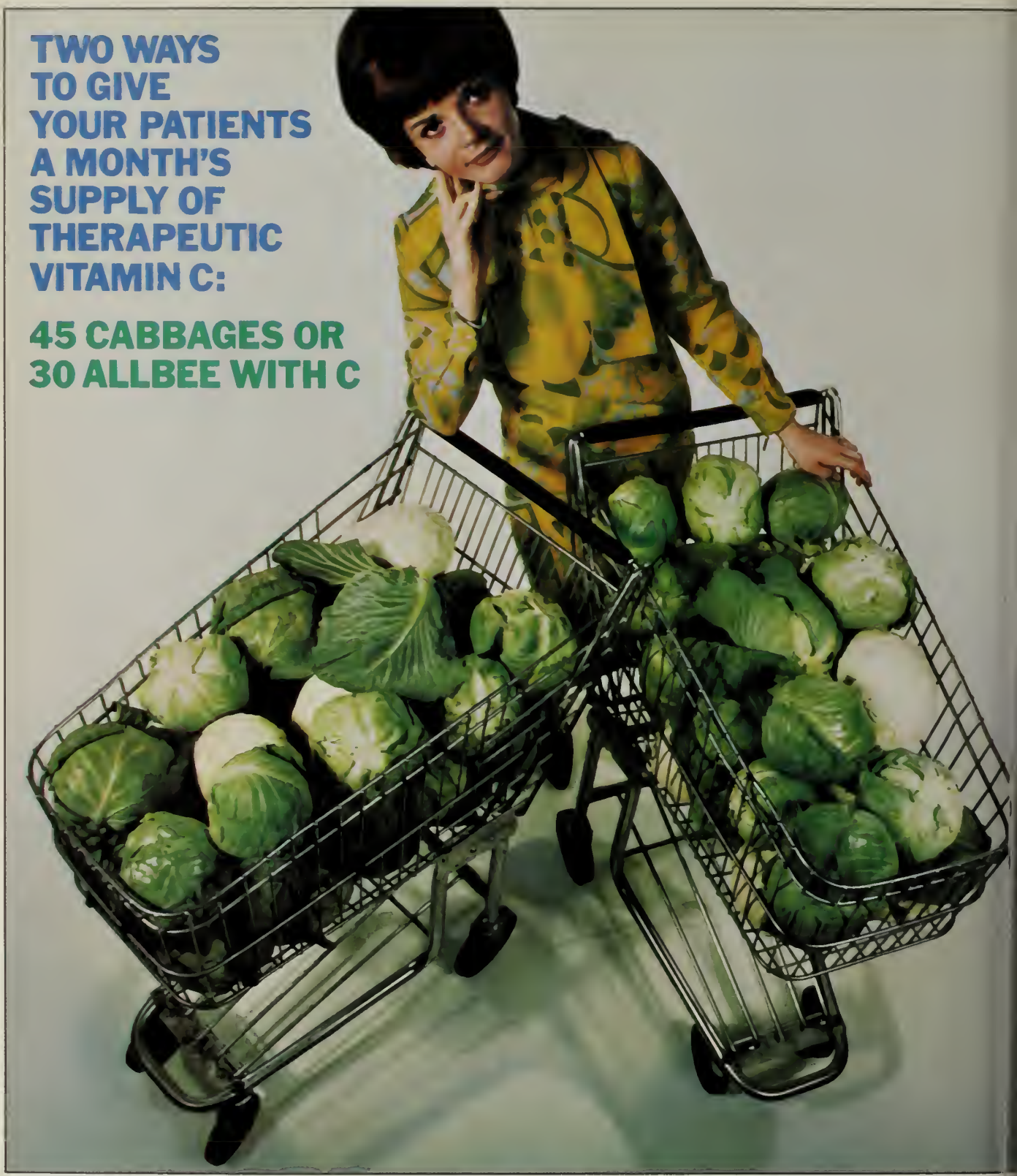
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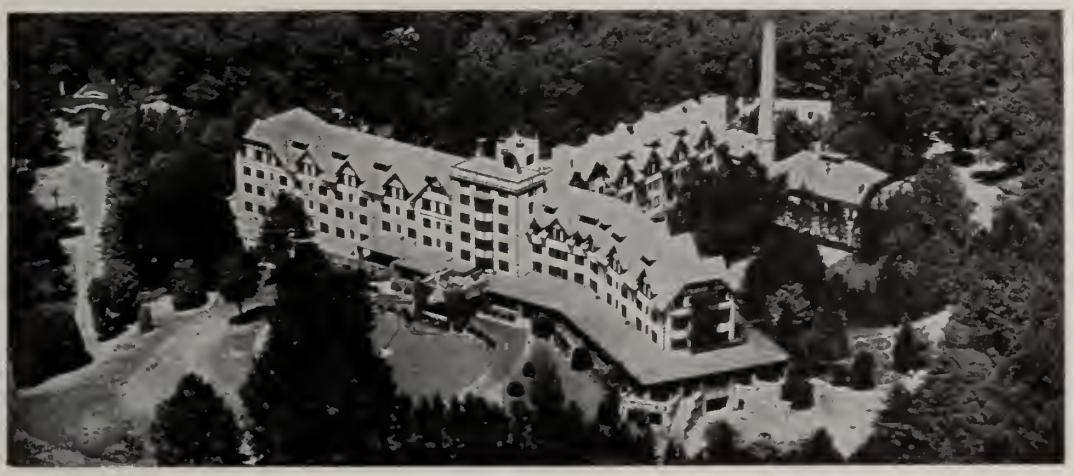
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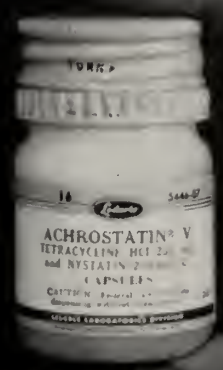
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Important Precautions: Carefully supervise dose and amounts prescribed, especially for patients prone to overdose themselves. Excessive prolonged use has been reported to result in dependence or habituation in susceptible persons, as alcoholics, ex-addicts, and other severe psychoneurotics. After prolonged excessive dosage, reduce dosage gradually to avoid possibly severe withdrawal reactions. Abrupt discontinuance of excessive doses has sometimes resulted in epileptiform seizures.

Warn patients of possible reduced alcohol tolerance, with resultant slowing of reaction time and impairment of judgment and coordination.

Reduce dose if drowsiness, ataxia or visual disturbance occurs; if persistent, patients should not operate vehicles or dangerous machinery.

Side Effects include drowsiness, usually transient; if persistent and associated with ataxia, usually responds to dose reduction; occasionally concomitant CNS stimulants (amphetamine, mephentermine sulfate) are desirable. Allergic or idiosyncratic reactions are rare, but such reactions, sometimes severe, can develop in patients receiving only 1 to 4 doses who have had no previous contact with meprobamate. Previous history of allergy may or may not be related to incidence of reactions. Mild reactions are characterized by itchy urticarial or erythematous maculopapular rash, generalized or confined to groin. Acute nonthrombocytopenic purpura with cutaneous petechiae, ecchymoses, peripheral edema and fever have been reported. One fatal case of bullous dermatitis following intermittent use of meprobamate with prednisolone has been reported. If allergic reaction occurs, meprobamate should be stopped and not reinstituted. Severe reactions, observed very rarely, include angioneurotic edema, bronchial spasms, fever, fainting spells, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case) and hyperthermia. Treat symptomatically as with epinephrine, antihistamine and possibly hydrocortisone. Aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis and hemolytic anemia have occurred rarely, almost always in presence of known toxic agents. A few cases of leukopenia, usually transient, have been reported on continuous administration.

Meprobamate may sometimes precipitate grand mal attacks in patients susceptible to both grand and petit mal. Extremely large doses can produce rhythmic fast activity in the cortical pattern. Impairment of accommodation and visual acuity has been reported rarely. After excessive dosage for weeks or months, withdraw gradually (1 or 2 weeks) to avoid recurrence of pretreatment symptoms (insomnia, severe anxiety, anorexia). Abrupt discontinuance of excessive doses has sometimes resulted in vomiting, ataxia, tremors, muscle twitching and epileptiform seizures. Prescribe very cautiously and in small amounts for patients with suicidal tendencies. Suicidal attempts have resulted in coma, shock, vasomotor and respiratory collapse and anuria. Excessive doses have resulted in prompt sleep; reduction of blood pressure, pulse and respiratory rates to basal levels; and occasionally hyperventilation. Treat with immediate gastric lavage and appropriate symptomatic therapy. (CNS stimulants and pressor amines as indicated.) Doses above 2400 mg./day are not recommended.

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When the talk turns to oral contraceptives, it makes medical sense to remember low-dose Norinyl®-1.

(norethindrone 1mg. & mestranol 0.05mg.)

Turn page for contraindications, precautions and side effects.



Reduction of oral contraceptive dosage to the lowest effective levels is a well-accepted principle of conservative medical practice. In keeping with this view, Norinyl is now also available as Norinyl-1, containing exactly one half the previous dosage of norethindrone and mestranol. Clinical experience has established that effective fertility control can be achieved with the same degree of reliability and safety with new Norinyl-1 when taken as directed.

What about switching patients from higher dosage forms?

In transferring patients to low-dose Norinyl-1 from higher-dosage oral contraceptives, some breakthrough bleeding may occur in the early cycles. In the majority of cases the bleeding episode is mild and self-limited. The long-term advantages of the lower dosage form should be weighed against the inconvenience of possible breakthrough bleeding in the individual patient.

Contraindications: 1. Patients with thrombophlebitis or with a history of thrombophlebitis or pulmonary embolism. 2. Liver dysfunction or disease. 3. Patients with known or suspected carcinoma of the breast or genital organs. 4. Undiagnosed vaginal bleeding.

Warnings: 1. Discontinue medication pending examination if there is sudden partial or complete loss of vision or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions, medication should be withdrawn. 2. Since the safety of NORINYL-1 (norethindrone 1 mg. with mestranol 0.05 mg.) in pregnancy has not been demonstrated, it is recommended that for any patient who has missed two consecutive periods, pregnancy should be ruled out before continuing the contraceptive regimen. If the patient has not adhered to the prescribed schedule, the possibility of pregnancy should be considered at the time of the first missed period. 3. Detectable amounts of the active ingredients in oral contraceptives have been identified in the milk of mothers receiving these drugs. The significance of this dose to the infant has not been determined.

Precautions: 1. The pretreatment physical examination should include special reference to breast and pelvic organs, as well as a Papanicolaou smear. 2. Endocrine and possibly liver function tests may be affected by treatment with NORINYL-1. Therefore, if such tests are abnormal in a patient taking NORINYL-1 it is recommended that they be repeated after the drug has been withdrawn for two months. 3. Under the influence of estrogen-progestogen preparations, preexisting uterine fibromyomata may increase in size. 4. Because these agents may cause some degree of fluid retention, conditions that may be influenced by this factor, such as epilepsy, migraine, asthma, cardiac or renal dysfunction, require careful observation. 5. NORINYL-1 should be used with caution in patients with a history of cerebrovascular accident. 6. In relation to breakthrough bleeding, and as in all cases of irregular bleeding *per vaginam*, nonfunctional causes should be borne in mind. In cases of undiagnosed vaginal bleeding, adequate diagnostic measures are indicated. 7. Patients with a history of psychic depression should be carefully observed and the drug discontinued if the depression recurs to a serious degree. 8. Any possible influence of prolonged NORINYL-1 therapy on pituitary, ovarian, adrenal, hepatic or uterine function awaits further study. 9. A decrease in glucose tolerance has been observed in a small percentage of patients on oral contraceptives. The mechanism of this decrease is obscure. For this reason, diabetic patients should be carefully observed while receiving NORINYL-1 therapy. 10. Because of the occasional occurrence of thrombophlebitis and pulmonary embolism in patients taking oral contraceptives, the physician should be alert to the earliest manifestations of the disease. 11. Because of the effects of estrogens on epiphyseal closure, NORINYL-1 should be used judiciously in young patients in whom bone growth is not complete. 12. The age of the patient constitutes no absolute limiting factor, although treatment with NORINYL-1 may mask the onset of the climacteric. 13. The pathologist should be advised of NORINYL-1 therapy when relevant specimens are submitted.

Side effects observed in patients receiving oral contraceptives: The following adverse reactions have been observed in patients receiving oral contraceptives: nausea, vomiting, gastrointestinal symptoms (such as abdominal cramps and bloating), breakthrough bleeding, spotting, change in menstrual flow, amenorrhea, edema, chloasma or melasma, breast changes (tenderness, enlargement and secretion), change in weigh (increase or decrease), changes in cervical erosion and cervical secretion suppression of lactation when given immediately postpartum, cholestatic jaundice, migraine, rash (allergic), rise in blood pressure in susceptible individuals, mental depression.

Although the following side effects have been reported in users of oral contraceptives, no cause and effect relationship has been established: anovulation post-treatment, premenstrual-like syndrome, change in libido, changes in appetite, cystitis like syndrome, headache, nervousne dizziness, fatigue, backache, hirsutism, loss of scalp hair, erythema multiforme, erythema nod sum, hemorrhagic eruption, itching

The following occurrences have bee observed in users of oral contraceptives (a cause and effect relationship has been neither established nor dis proved): thrombophlebitis, pulmona embolism, neuro-ocular lesions.

The following laboratory results may be altered by the use of oral contraceptives: increased sulfobromophthalein and other hepatic function tests, coagulation tests (increase in prothrombin, Factors VII, VIII, IX and X), thyroid function (increase in PBI and butanol extractable protein-bound iodine an decrease in T^3 values), metyrapone test, pregnanediol determination.

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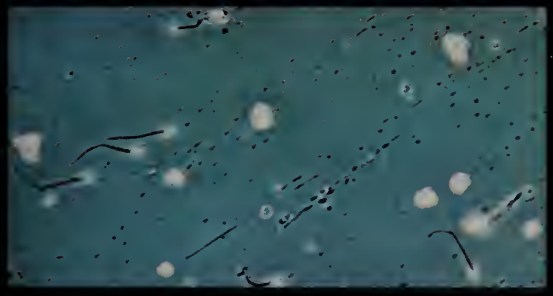
Here's why Norinyl-1 makes medical sense.

The effectiveness of Norinyl-1 as a low-dose oral contraceptive may be explained by its possible multiple action. In addition to its primary action of suppression of ovulation, Norinyl-1 may offer additional protective mechanisms... (1) creation of a cervical mucus that may be hostile to sperm penetration, and (2) development of an endometrium that may be out of phase with nidation. These effects are illustrated below.

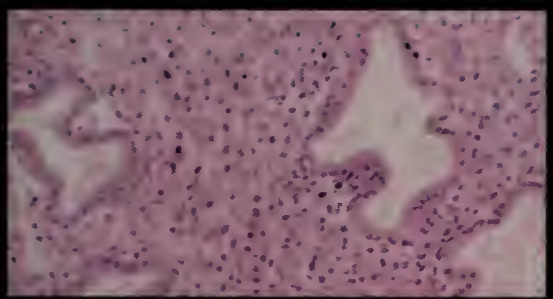
Untreated Patient



Cervical mucus at midcycle is usually thin and watery, with Spinnbarkeit (stretchability) of 15 to 20 cm.



Spermatozoa appear healthy, active, freemoving.

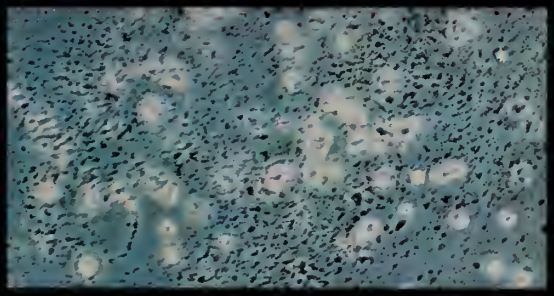


Endometrium of untreated patient is receptive to the fertilized ovum during secretory phase.

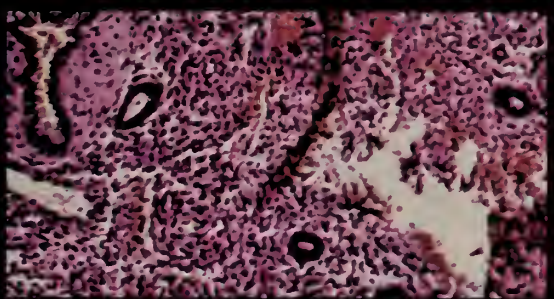
Norinyl-1 Patient



Cervical mucus at midcycle is scanty, viscous—with Spinnbarkeit of 1 cm. or less.



Immobile spermatozoa as they appear in cervical mucus taken from patient treated with Norinyl-1.



Norethindrone in Norinyl-1 accelerates secretory phase, suppresses glandular and vascular development.

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Warning: With the administration of enteric-coated potassium supplements, which should be used only when adequate dietary supplementation is not practical, the possibility of small bowel lesions (obstruction, hemorrhage, and perforation) should be kept in mind. Surgery for these lesions has frequently been required and deaths have occurred. Discontinue enteric-coated potassium supplements immediately if abdominal pain, distention, nausea, vomiting, or gastrointestinal bleeding occur. Use with caution in pregnant patients, since the drug may cross the placental barrier and adverse reactions which may occur in the adult (thrombocytopenia, hyperbilirubinemia, altered carbohydrate metabolism, etc.) are potential problems in the newborn.
Precautions: Antihypertensive therapy with Hygroton should always be initiated cautiously in postsympathectomy patients and in patients receiving ganglionic blocking agents or other potent antihypertensive drugs, or curare. Reduce dosage of concomitant antihypertensive agents by at least one-half. Barbiturates, narcotics or alcohol may potentiate hypotension. Be
cause of the possibility of progr
sion of renal damage, periodic
determination of the BUN is ind
cated. Discontinue if the BUN r
or liver dysfunction is aggravat
Hepatic coma may be precipita
Electrolyte imbalance, sodium
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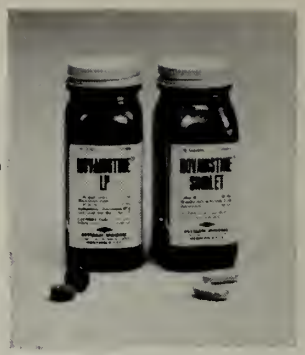
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Whether you prescribe Novahistine LP or Novahistine Singlet, a total daily dose of 3 or 4 tablets will usually provide effective, continuous relief.
Use cautiously in patients with severe hypertension, diabetes mellitus, hyperthyroidism or urinary retention. Caution ambulatory patients that drowsiness may result.

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Please see Brief Summary on next page.

*Clinically cured included patients with complete symptomatic relief and those who had partial symptomatic relief within 72 hours with subsequent clinical cure.

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Indications For mixed infections which are more susceptible to the combination than to either agent alone. These may include susceptible gram-positive and/or gram-negative mixed infections. Not recommended in the treatment of infections expected to respond to full therapeutic doses of the antibiotic or sulfonamide alone.

Contraindications Known hypersensitivity to erythromycin or sulfonamides. Because of the possibility of kernicterus with sulfonamides, do not use in pregnancy at term, in premature or in newborn infants during first week of life.

Warnings As with other forms of sulfonamide therapy, use only after critical appraisal in patients with liver or kidney damage, urinary obstruction, or blood dyscrasias. Deaths have been reported from hypersensitivity reactions and blood dyscrasias following use of sulfonamides. Perform blood counts and liver and kidney function tests when used intermittently or for long periods.

Precautions, Side Effects Use sulfonamides with caution in patients with a history of allergy, including asthma. Mild allergic reactions (such as urticaria and other skin rashes) may occur. Serious allergic reactions have been extremely infrequent; if encountered appropriate countermeasures (e.g. epinephrine, steroids, etc.) should be administered and the drug withdrawn. Assure adequate fluid intake to prevent crystalluria and institute alkali therapy if indicated. In prolonged therapy, or when high dosage is used, maintain an alkaline urine. If oliguria occurs, discontinue therapy. Occasionally mild abdominal discomfort, nausea or vomiting may occur with erythromycin; generally improved by reduction of dosage.

If overgrowth of nonsusceptible organism occurs, withdraw the drug and institute appropriate treatment.

Adverse Reactions Sulfonamide therapy may be associated with headache, nausea, vomiting, diarrhea, hepatitis, pancreatitis, blood dyscrasias, neuropathy, drug fever, skin rash, urticaria, injection of the conjunctiva and sclera, petechiae, purpura, hematuria and crystalluria. 803446

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* anticostive, *adj.* (*anti* opposed to + *costive* causing constipation.) Against constipation. (Now isn't that a good idea in an iron-containing hematinic? We'll send you samples if you'll send a request on your Rx blank, addressed to Department 150.)

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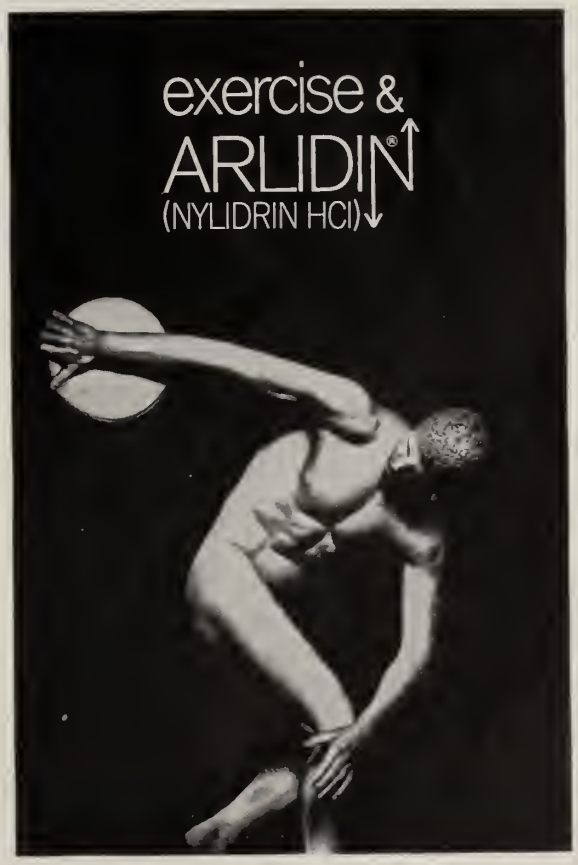
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1. Bradley, J. E., *et al.*: J. Pediat. *38*:41 (Jan.) 1951.
2. Bradley, J. E.: Mod. Med. *20*:71 (Oct. 15) 1952.
3. Crunden, A. B., Jr., and Davis, W. A.: Am. J. Obst. & Gynec. *65*:311 (Feb.) 1953.



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Warnings: Not of value in treatment of psychotic patients, and should not be employed in lieu of appropriate treatment. As with most CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may also be associated with temporary increase in frequency and/or severity of seizures. Advise patients against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance. Keep addiction-prone individuals (such as drug addicts or alcoholics) under careful surveillance because of their predisposition to habituation and dependence. Use of any drug in pregnancy, lactation or in women of childbearing age requires that potential benefit be weighed against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects—particularly with known compounds which may potentiate action of Valium, such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants. Employ usual precautions in the severely depressed or in those with latent depression; suicidal tendencies may be present and protective measures necessary. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed or tolerated).

Adverse Reactions: Side effects most commonly reported: drowsiness, fatigue and ataxia. Infrequently encountered: confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo and blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, use of the drug should be discontinued. Because of isolated reports of neutropenia and jaundice, periodic blood counts and liver function tests are advisable during long-term therapy. Minor changes in EEG patterns (low-voltage fast activity) observed during and after therapy and are of no known significance.

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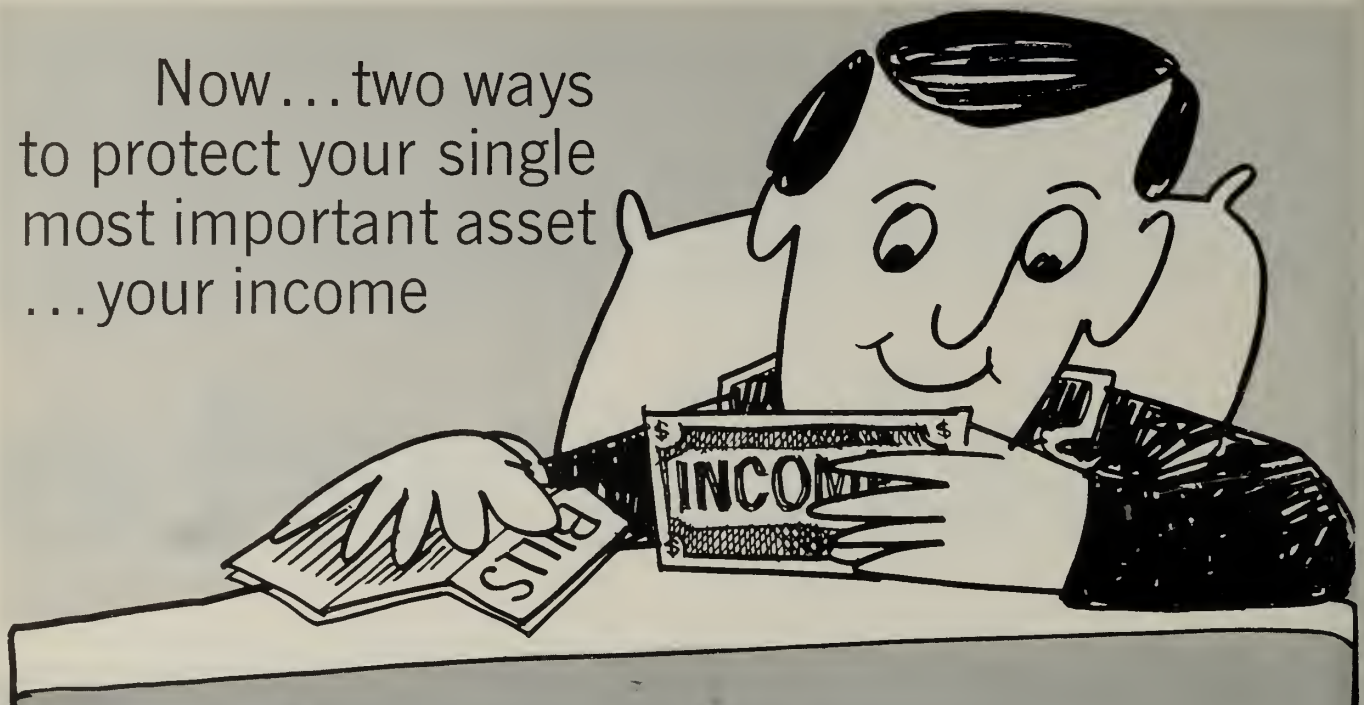
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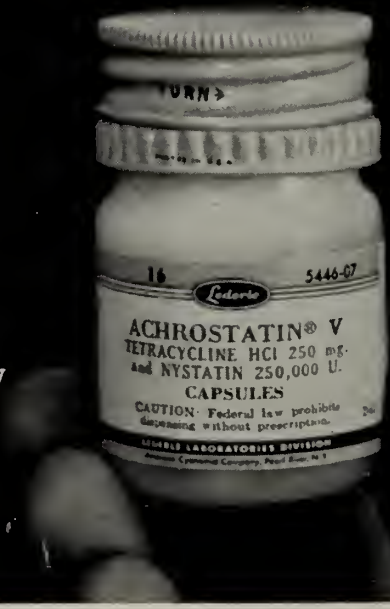
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In transferring patients to low-dose Norinyl-1 from higher-dosage oral contraceptives, some breakthrough bleeding may occur in the early cycles. In the majority of cases the bleeding episode is mild and self-limited. The long-term advantages of the lower dosage form should be weighed against the inconvenience of possible breakthrough bleeding in the individual patient.

Contraindications: 1. Patients with thrombophlebitis or with a history of thrombophlebitis or pulmonary embolism. 2. Liver dysfunction or disease. 3. Patients with known or suspected carcinoma of the breast or genital organs. 4. Undiagnosed vaginal bleeding.

Warnings: 1. Discontinue medication pending examination if there is sudden partial or complete loss of vision or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions, medication should be withdrawn. 2. Since the safety of NORINYL-1 (norethindrone 1 mg. with mestranol 0.05 mg.) in pregnancy has not been demonstrated, it is recommended that for any patient who has missed two consecutive periods, pregnancy should be ruled out before continuing the contraceptive regimen. If the patient has not adhered to the prescribed schedule, the possibility of pregnancy should be considered at the time of the first missed period. 3. Detectable amounts of the active ingredients in oral contraceptives have been identified in the milk of mothers receiving these drugs. The significance of this dose to the infant has not been determined.

Precautions: 1. The pretreatment physical examination should include special reference to breast and pelvic organs, as well as a Papanicolaou smear. 2. Endocrine and possibly liver function tests may be affected by treatment with NORINYL-1. Therefore, if such tests are abnormal in a patient taking NORINYL-1 it is recommended that they be repeated after the drug has been withdrawn for two months. 3. Under the influence of estrogen-progestogen preparations, preexisting uterine fibromyomata may increase in size. 4. Because these agents may cause some degree of fluid retention, conditions that may be influenced by this factor, such as epilepsy, migraine, asthma, cardiac or renal dysfunction, require careful observation. 5. NORINYL-1 should be used with caution in patients with a history of cerebrovascular accident. 6. In relation to breakthrough bleeding, and as in all cases of irregular bleeding *per vaginam*, nonfunctional causes should be borne in mind. In cases of undiagnosed vaginal bleeding, adequate diagnostic measures are indicated. 7. Patients with a history of psychic depression should be carefully observed and the drug discontinued if the depression recurs to a serious degree. 8. Any possible influence of prolonged NORINYL-1 therapy on pituitary, ovarian, adrenal, hepatic or uterine function awaits further study. 9. A decrease in glucose tolerance has been observed in a small percentage of patients on oral contraceptives. The mechanism of this decrease is obscure. For this reason, diabetic patients should be carefully observed while receiving NORINYL-1 therapy. 10. Because of the occasional occurrence of thrombophlebitis and pulmonary embolism in patients taking oral contraceptives, the physician should be alert to the earliest manifestations of the disease. 11. Because of the effects of estrogens on epiphyseal closure, NORINYL-1 should be used judiciously in young patients in whom bone growth is not complete. 12. The age of the patient constitutes no absolute limiting factor, although treatment with NORINYL-1 may mask the onset of the climacteric. 13. The pathologist should be advised of NORINYL-1 therapy when relevant specimens are submitted.

Side effects observed in patients receiving oral contraceptives: The following adverse reactions have been observed in patients receiving oral contraceptives: nausea, vomiting, gastrointestinal symptoms (such as abdominal cramps and bloating), breakthrough bleeding, spotting, change in menstrual flow, amenorrhea, edema, chloasma or melasma, breast changes (tenderness, enlargement and secretion), change in weight (increase or decrease), changes in cervical erosion and cervical secretion, suppression of lactation when given immediately postpartum, cholestatic jaundice, migraine, rash (allergic), rise in blood pressure in susceptible individuals, mental depression.

Although the following side effects have been reported in users of oral contraceptives, no cause and effect relationship has been established: anovulation post-treatment, premenstrual-like syndrome, change in libido, changes in appetite, cystitis-like syndrome, headache, nervousness, dizziness, fatigue, backache, hirsutism, loss of scalp hair, erythema multiforme, erythema nodosum, hemorrhagic eruption, itching.

The following occurrences have been observed in users of oral contraceptives (a cause and effect relationship has been neither established nor disproved): thrombophlebitis, pulmonary embolism, neuro-ocular lesions.

The following laboratory results may be altered by the use of oral contraceptives: increased sulfobromophthalein and other hepatic function tests, coagulation tests (increase in prothrombin, Factors VII, VIII, IX and X), thyroid function (increase in PBI and butanol extractable protein-bound iodine and decrease in T^3 values), metyrapone test, pregnanediol determination.

norethindrone — an original steroid from
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LABORATORIES INC., PALO ALTO, CALIF

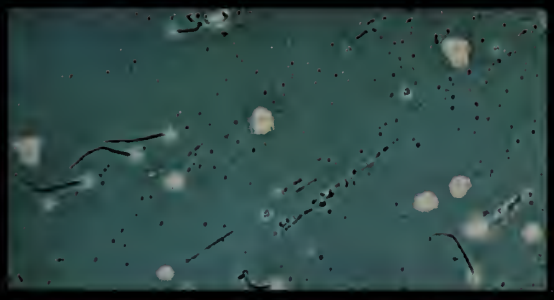
Here's why Norinyl-1 makes medical sense.

The effectiveness of Norinyl-1 as a low-dose oral contraceptive may be explained by its possible multiple action. In addition to its primary action of suppression of ovulation, Norinyl-1 may offer additional protective mechanisms... (1) creation of a cervical mucus that may be hostile to sperm penetration, and (2) development of an endometrium that may be out of phase with nidation. These effects are illustrated below.

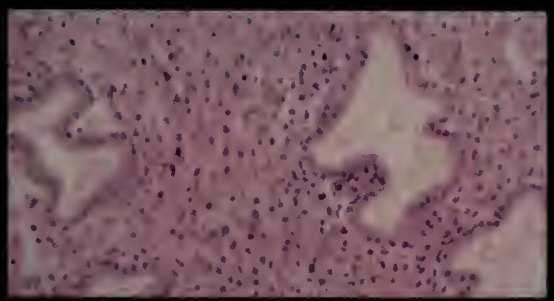
Untreated Patient



Cervical mucus at midcycle is usually thin and watery, with Spinnbarkeit (stretchability) of 15 to 20 cm.



Spermatozoa appear healthy, active, freemoving.

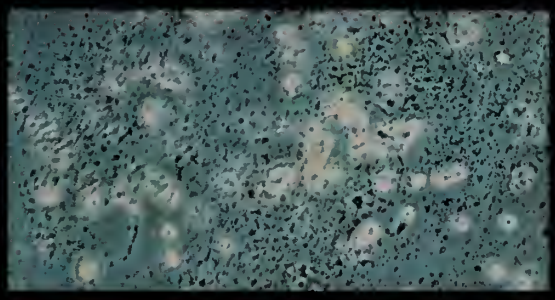


Endometrium of untreated patient is receptive to the fertilized ovum during secretory phase.

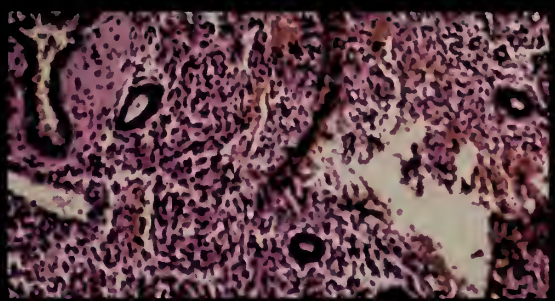
Norinyl-1 Patient



Cervical mucus at midcycle is scanty, viscous—with Spinnbarkeit of 1 cm. or less.



Immobile spermatozoa as they appear in cervical mucus taken from patient treated with Norinyl-1.



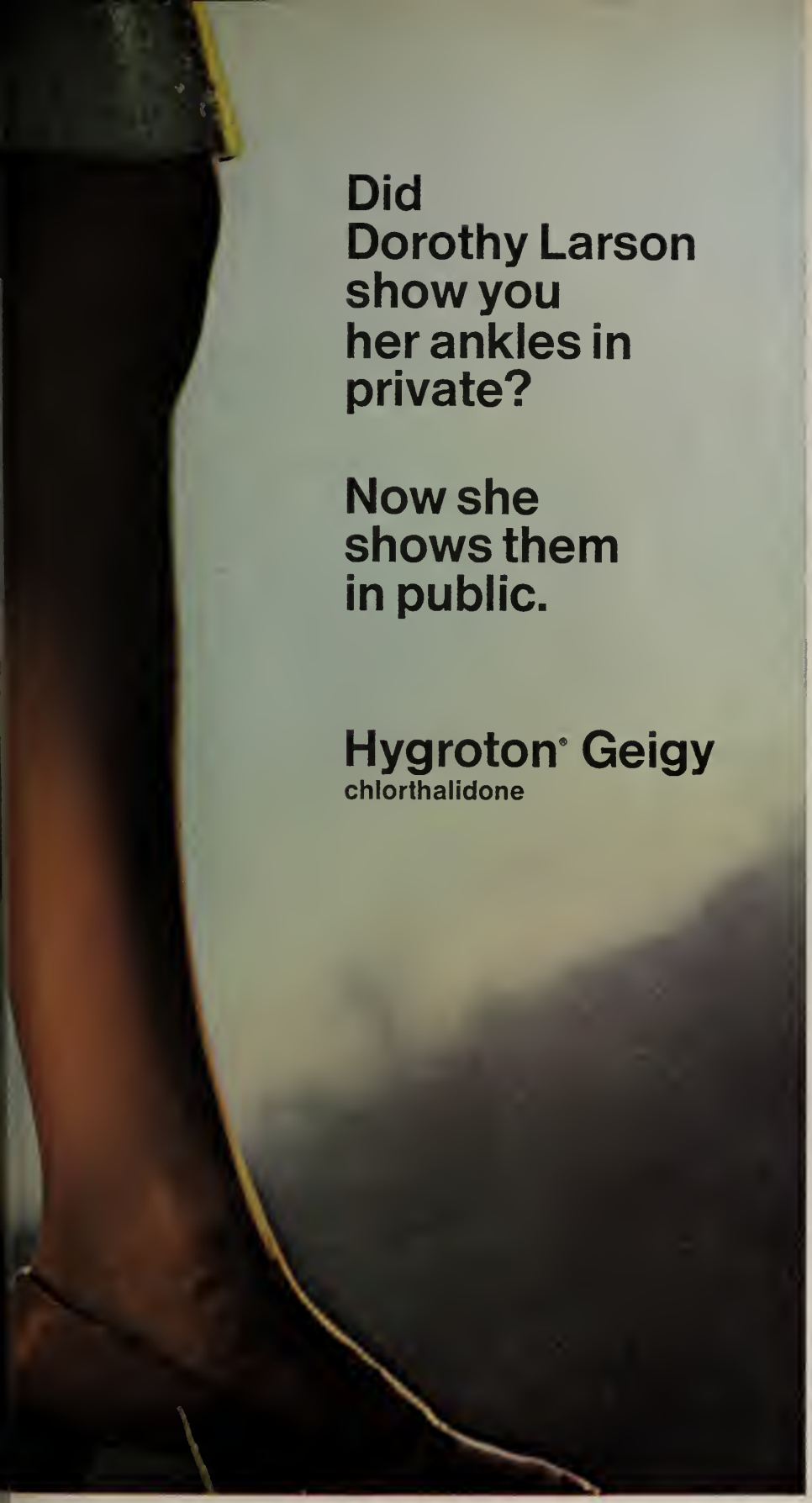
Norethindrone in Norinyl-1 accelerates secretory phase, suppresses glandular and vascular development.

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(norethindrone 1mg ē mestranol 0.05mg)

- new low dose of time-proved ingredients
- established norethindrone/mestranol ratio
- lower patient cost



Indications: Hypertension and many types of edema involving retention of salt and water.
Contraindications: Hypersensitivity and most cases of severe renal or hepatic disease.
Warning: With the administration of enteric-coated potassium supplements, which should be used only when adequate dietary supplementation is not practical, the possibility of small bowel lesions (obstruction, hemorrhage, and perforation) should be kept in mind. Surgery for these lesions has frequently been required and deaths have occurred. Discontinue enteric-coated potassium supplements immediately if abdominal pain, distention, nausea, vomiting, or gastrointestinal bleeding occur. Use with caution in pregnant patients, since the drug may cross the placental barrier and adverse reactions which may occur in the adult (thrombocytopenia, hyperbilirubinemia, altered carbohydrate metabolism, etc.) are potential problems in the newborn.
Precautions: Antihypertensive therapy with Hygroton should always be initiated cautiously in postsympathectomy patients and in patients receiving ganglionic blocking agents or other potent antihypertensive drugs, or curare. Reduce dosage of concomitant antihypertensive agents by at least one-half. Barbiturates, narcotics or alcohol may potentiate hypotension. Because of the possibility of progr sion of renal damage, periodic determination of the BUN is indi cated. Discontinue if the BUN r or liver dysfunction is aggravate Hepatic coma may be precipitat Electrolyte imbalance, sodium a or potassium depletion may occ If potassium depletion should o cur during therapy, Hygroton sh be discontinued and potassium supplements given, provided the



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Your office examination confirmed Mrs. Larson's ankle edema.

You prescribed Hygroton to get rid of the edema.

And you found that Hygroton is not only usually effective; it frequently costs less than other equivalent therapy.

A nice way to treat the Mrs. Larsons in your practice.

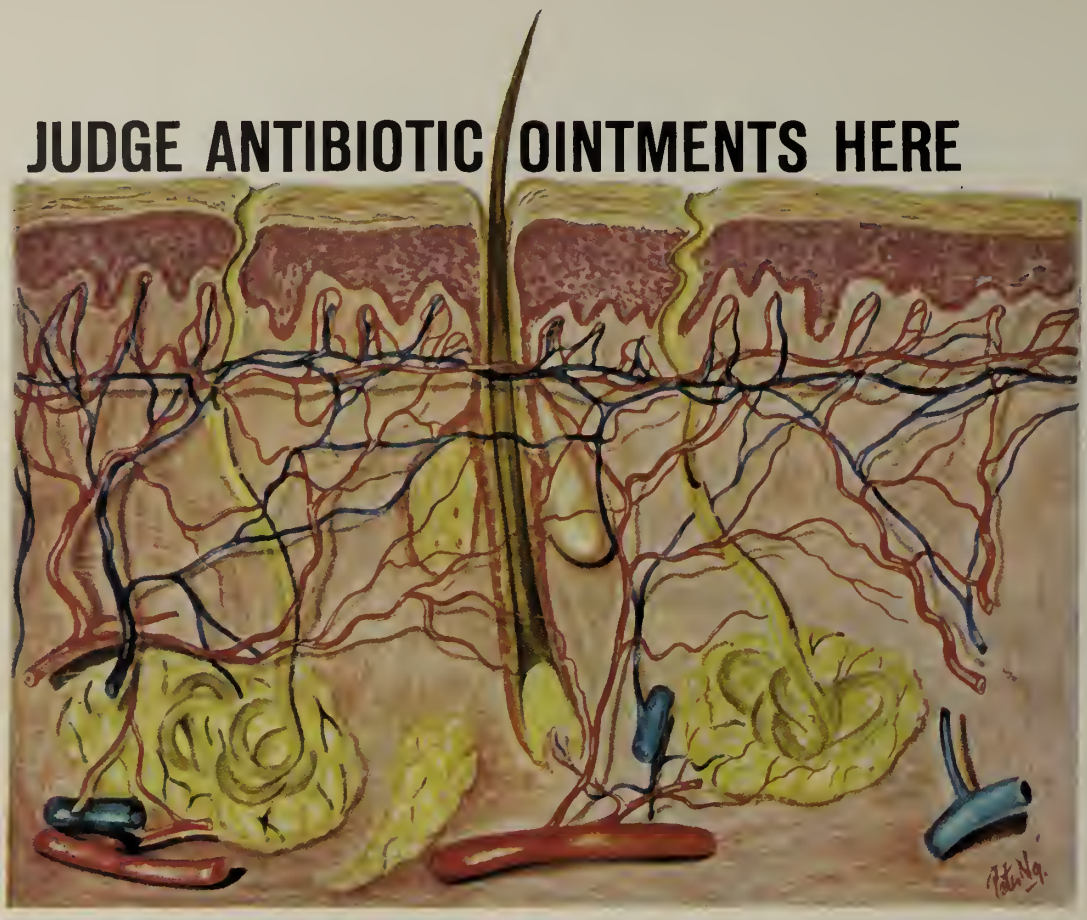
Hygroton therapy may mean troublesome side effects for some patients. And you can't prescribe it for patients with hypersensitivity to the drug or severe renal or hepatic diseases. Before writing it for your patients, please check the prescribing information. It's summarized below.

ient does not have marked oli-
ia.
e special care in cirrhosis or
ere ischemic heart disease and
atients receiving corticoste-
ds, ACTH, or digitalis. Salt re-
ction is not recommended.
erse Reactions: Nausea, gastric
ation, vomiting, anorexia, con-
ation and cramping, dizziness,
kness, restlessness, hypergly-
ia, hyperuricemia, headache,
cle cramps, orthostatic hypo-
tension, aplastic anemia, leuko-
penia, thrombocytopenia, agranu-
locytosis, impotence, dysuria,
transient myopia, skin rashes, urti-
caria, purpura, necrotizing angiitis,
acute gout, and pancreatitis when
epigastric pain or unexplained G.I.
symptoms develop after prolonged
administration. Other reactions re-
ported with this class of compounds
include: jaundice, xanthopsia,
paresthesia, and photosensitization.
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other day.
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Lederle

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Parepectolin for quick relief of acute diarrhea
... soothes colicky pain with paregoric*
... consolidates fluid stools with pectin
... adsorbs irritants with kaolin,
and protects intestinal mucosa

In children, Parepectolin may be used to control diarrhea promptly and prevent dehydration, until etiology has been determined. In some cases, Parepectolin may be all the therapy necessary.

Parepectolin®

Each fluid ounce of creamy white suspension contains:
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Contains opium (¼ grain) 15 mg. per fluid ounce.
warning: may be habit forming
Pectin ..... (2½ grains) 162 mg.
Kaolin (specially purified) .... (85 grains) 5.5 Gm.
(alcohol 0.69%)
Usual Children's Dose: One or two teaspoonfuls three times daily.

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of Peritinic

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* anticostive, *adj.* (*anti* opposed to + *costive* causing constipation.) Against constipation. (Now isn't that a good idea in an iron-containing hematinic?)

PERITINIC®

Hematinic with Vitamins and Fecal Softener

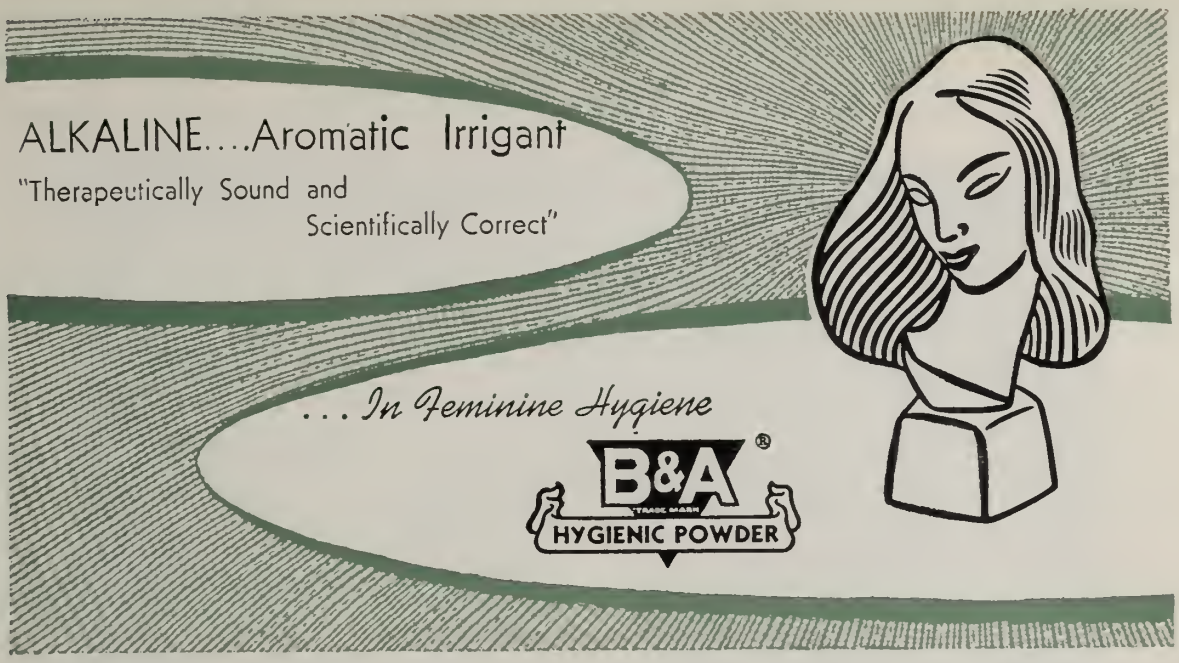
A tablet-a-day provides:

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Vitamin B_1	7.5 mg
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Vitamin B_6	7.5 mg
Vitamin B_{12}	50 mcgm
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Folic Acid	0.05 mg
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Bottles of 60

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489-7—6063



"Early in 1953 Davis and Grand undertook a review of well established practices connected with the treatment of T. vaginalis infections. For instance, it had been accepted that an acid douche should be employed. This was based on cultural studies showing that when the pH was below 5 the organisms would die. They found that the commonly used vinegar douche with a pH of 3 showed no visible effect on T. vaginalis under the microscope in 15 minutes. Both citric and lactic acid were tested with similar results. Davis then recalled that in his early treatment of T. Vaginalis infections over 30 years ago he had had his first successes with alkaline preparations, and when he returned to their use the patients reported them more soothing than the previously used acid solutions."

(Am. J. Obst. & Gynec., 68:559, 1954)

Criteria for Cleansing Douche

"Daily douching often is a part of the treatment administered in dealing with T. vaginalis and since it is possible that the solution might contain agents that could hamper the regrowth of Doederlein bacillus, the following properties were said to be ideal.

1. It should contain a harmless, nonsensitizing detergent to remove mucous deposits and debris.
2. It should not contain any antibacterial agents, since the contact possible during douching would be so fleeting that anti-bacterial action could not be expected.
3. It should be acid in pH.

The 3rd property seems to be the most illogical and unimportant. If contact is so fleeting that antibacterial actions could not be expected, it would seem to follow that its acid quality would be of no consequence. Furthermore, acid solutions are irritating to the mucosa and oftentimes accentuate inflammation.

Actually, the value of a douche is generally conceded to be confined to its use as a cleansing and deodorizing agent. The encouraging results achieved with a detergent douche solution in treating infections of the vagina are probably attributable to the enormous normal recuperative powers of the vaginal tissues which have been under-estimated as a factor in the restoration of normal physiology.

Therefore, simple cleansing of the vagina with a non-irritating, mildly alkaline douche which is soothing to the vaginal mucosa and which penetrates and flushes out the dead organisms, debris and mucinous materials frequently enables the physiological processes to overcome the infection."

Charles B. Marek, M.D., Chief of Gynecology; Bon Secours Hospital: personal communications August 11, 1964

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Contraindications: Comatose states or the presence of large amounts of C.N.S. depressants.

Precautions: Potentiation of C.N.S. depressants may occur (reduce dosage of such agents when used concomitantly). Use with caution in patients with chronic respiratory disorders. Antiemetic effect may mask overdosage of toxic drugs or obscure other conditions. Administer in pregnancy only when necessary. Because of possible drowsiness use cautiously and warn patients who operate vehicles or machinery.

Adverse Reactions: Drowsiness; dry mouth; nasal congestion; constipation; amenorrhea; miosis; mild fever; weight gain; hypotensive effects, sometimes severe with I.M. administration; epinephrine effects may be reversed; dermatological reactions; parkinsonism-like symptoms on high dosages (in rare instances, may persist); lactation and moderate breast engorgement (in females on high dosages); and less frequently, cholestatic jaundice (use cautiously in patients with liver disease). Adverse reactions occurring rarely, include: mydriasis; agranulocytosis; skin pigmentation; epithelial keratopathy; lenticular and corneal deposits (after prolonged substantial doses).

Available: Tablets, 10 mg., 25 mg., 50 mg., 100 mg. and 200 mg.; Spansule® capsules, 30 mg., 75 mg., 150 mg., 200 mg. and 300 mg.; Injection, 25 mg./cc.; Syrup, 10 mg./5 cc.; Suppositories, 25 mg. and 100 mg.

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Relieve painful skeletal muscle spasm in your office in minutes with a single 2 cc. injection of NORFLEX. Then, for sustained relief, write a prescription for NORFLEX tablets, 1 tablet b.i.d.

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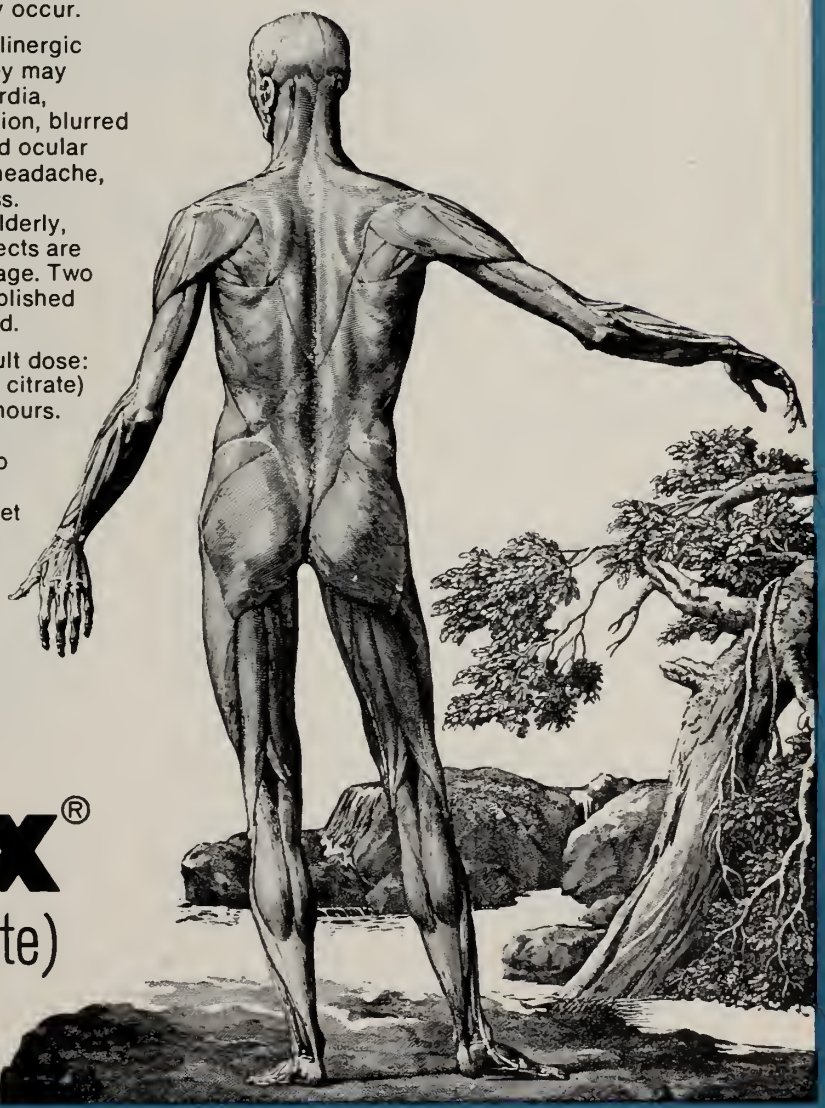
WARNING: Transient lightheadedness or dizziness following NORFLEX-INJECTABLE may occur.

SIDE EFFECTS: Due mainly to anticholinergic action and usually at high dosage. They may include dryness of the mouth, tachycardia, palpitation, urinary hesitancy or retention, blurred vision, dilatation of the pupil, increased ocular tension, weakness, nausea, vomiting, headache, dizziness, constipation, and drowsiness. Infrequently, mental confusion in the elderly, urticaria or other dermatoses. Side effects are usually eliminated by reduction in dosage. Two cases of aplastic anemia, with no established causal relationship, have been reported.

DOSAGE: **INJECTABLE** — Average adult dose: one ampul, 2 cc. (60 mg. orphenadrine citrate) I.M. or I.V. May be repeated every 12 hours. Relief may be maintained with one NORFLEX tablet b.i.d. **TABLETS** — Two tablets per day for adults, one in the morning, one in the evening. Each tablet contains 100 mg. orphenadrine citrate.

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"For weeks I've done practically nothing and I'm always tired. I wake up tired and I go to bed tired. It's really absurd."

When the patient complains of fatigue, and you can find no organic cause, you recognize that it may serve her as a means of avoiding responsibilities or facing an emotional problem. It is, in effect, a psychological retreat behind a somatic cover of continuous fatigue —one of the many depressive symptoms often associated with psychic tension.

She needs counsel and reassurance, and perhaps a tranquilizer to attenuate excessive tension and help restore the capacity to cope. As an aid to successful management, consider the value of Valium® (diazepam). As psychic tension is eased by Valium therapy, secondary depressive symptoms too may subside. The patient feels more capable, therefore more hopeful; better able to handle situations of intense stress.



Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in: skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders; athetosis, stiff-man syndrome, convulsive disorders (not for sole therapy).

Contraindications: Known hypersensitivity to drug; children under 6 months of age; acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in treatment of psychotic patients, and should not be employed in lieu of appropriate treatment. As with most CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may also be associated with temporary increase in frequency and/or severity of seizures. Advise patients against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance. Keep addiction-prone individuals (such as drug addicts or alcoholics) under careful surveillance because of their predisposition to habituation and dependence. Use of any drug in pregnancy, expectation or in women of childbearing age requires that potential benefit be weighed against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects particularly with known compounds which may potentiate action of Valium, such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants. Employ usual precautions in the severely depressed or in those with latent depression; suicidal tendencies may be present and protective measures necessary. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed or tolerated).

Adverse Reactions: Side effects most commonly reported: drowsiness, fatigue and ataxia. Infrequently encountered: confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo and blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, use of the drug should be discontinued. Because of isolated reports of neutropenia and jaundice, periodic blood counts and liver function tests are advisable during long-term therapy. Minor changes in EEG patterns (low-voltage fast activity) observed during and after therapy and are of no known significance.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See **Precautions**.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® (diazepam) Tablets, 2 mg, 5 mg, and 10 mg; bottles of 50, 100 and 500.

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Valium® (diazepam)

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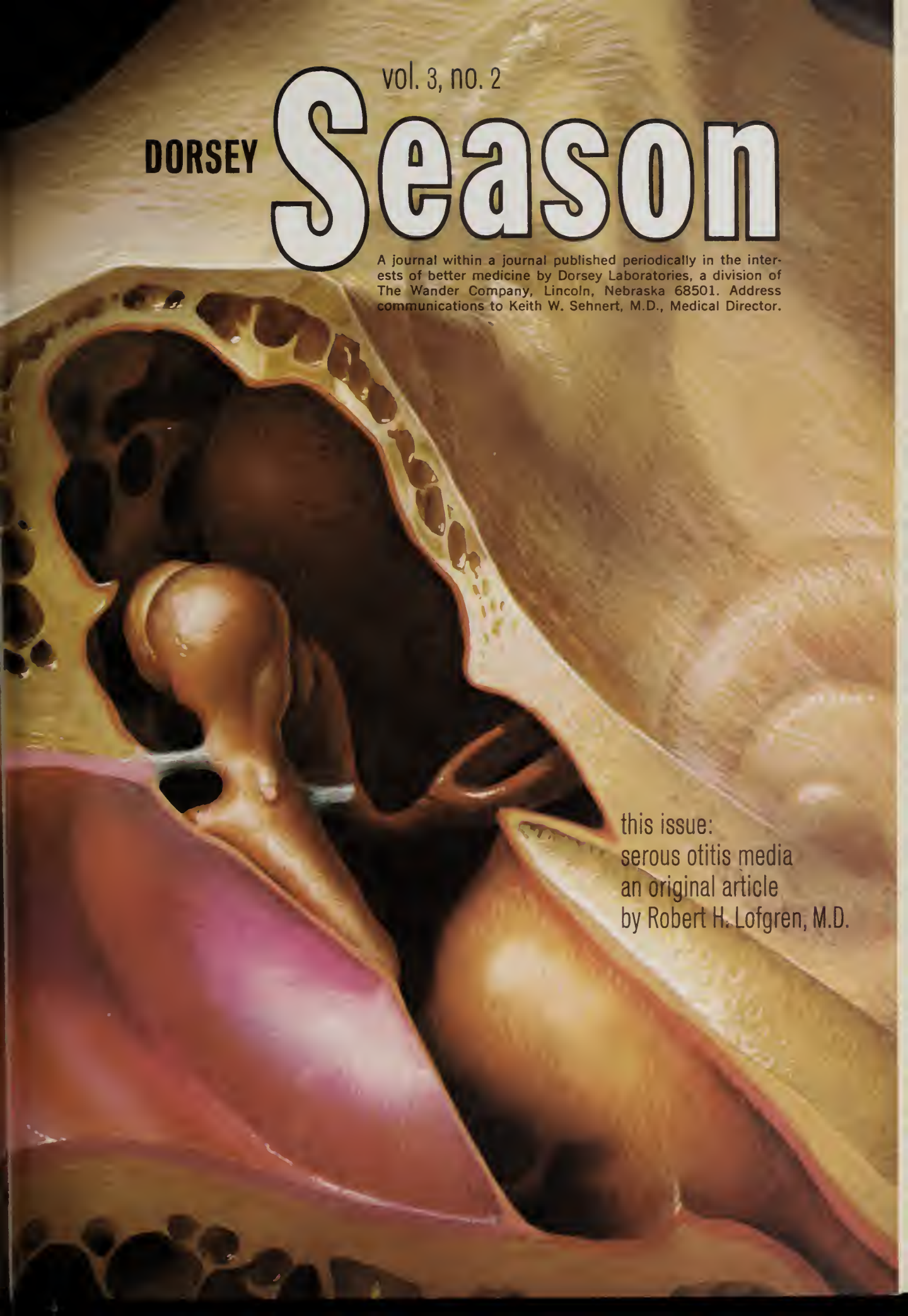
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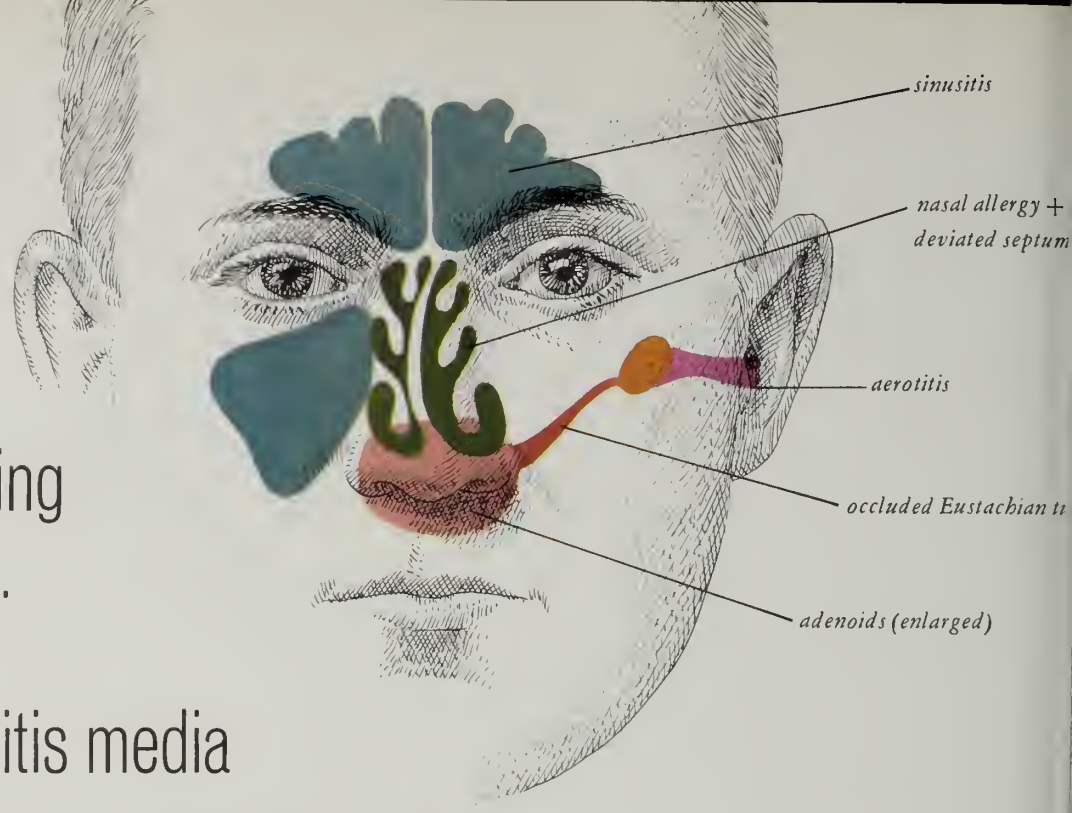
vol. 3, no. 2

DORSEY Season

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this issue:
serous otitis media
an original article
by Robert H. Lofgren, M.D.



the complaining earache... key to serous otitis media

Robert H. Lofgren, M.D.
Associate Surgeon, Massachusetts Eye and Ear Infirmary, Boston, Massachusetts

Serous otitis media is probably the most common ear problem in children, especially in the wintertime. Diagnosis is usually easy in adults, but may be quite difficult in children, especially the very young child where one has to depend almost entirely on the physical findings.

The symptoms of serous otitis are a fullness in the ear, a mild hearing loss and mild earache. The earache can best be described as a "complaining earache" instead of a "screaming earache" as found in purulent otitis media. A young child may be irritable and pull at the ear. In milder cases, and as the ear recovers, gurgling and popping noises can be heard in the ear.

On examination one may see a yellow tympanic membrane and if a fluid level is present the diagnosis becomes easy. However, the eardrum may be dull gray, or slightly pink, or even perfectly normal. Pneumomassage using a Siegle otoscope or a closed-head electric otoscope must be done on both ears, otherwise the diagnosis will be frequently missed. Attempted movement of the drum with the pneumatic otoscope produces either a sluggish motion of the drum or no motion at all instead of the easily movable normal drum. The Rinne test is negative and there is a 15-20 decibel conductive hearing loss. The bone conduction may be better than normal and an air-bone gap may exist even with the air conduction within normal limits. Occasionally one finds a false nerve deafness on the audiogram when thick glue-like fluid causes immobility of both the oval and round window. Both conditions return to normal when the fluid is removed, but they make the interpretation of screening audiograms very difficult.

There is no single cause for serous otitis. One factor always present is blockage of the eustachian tube, but this alone is not enough to produce fluid. There must also be an inflammatory reaction. Blockage of the eustachian tube may be caused by many conditions. In children the most common cause is enlarged adenoids. In the summer the next most common cause is allergy. Upper respiratory infections or influenza are common causes in the winter. Nasal allergy, acute and chronic sinusitis, nasal septal deformity and cleft palate can all cause eustachian tube obstruction. Some children may have a congenitally small eustachian tube, but fortunately they usually "grow out of the problem."

The first sign of a nasopharyngeal tumor is often a serous otitis. One must always rule this out in any adult who later in life develops repeated or persisting serous otitis. Causes sometimes overlooked are nasogastric tubes after surgery, simple obesity and cardiorenal disease, which may produce congestion in the mucosal lining of the eustachian tube. In recent years we have been seeing a new cause—acute otitis media, where the patient is adequately treated with antibiotics but where drainage has not been

established either through the eardrum or down the eustachian tube. A sterile exudate is left in the middle ear.

The inflammatory response may be caused by a marked negative pressure as in air otitis from flying, or it may be from a mild bacterial or viral infection in the middle ear. Serous fluid is a good culture medium and will frequently go on to purulent otitis media, especially if the original blockage was caused by an infectious process such as acute rhinitis or adenoiditis. When the infection heals there may be scarring in the middle ear mucosa. Mucous glands develop in this tissue and pour out a thick mucoid material. This ear usually looks normal until a pneumatic otoscope is used. The objectives in treating serous otitis are to remove the obstructing agent and to provide drainage from the middle ear. Often this can be accomplished by decongestants and nose drops. If large obstructing adenoids are present they should be removed. Sinusitis should be treated with oral decongestants or nose drops, plus antibiotics where indicated. Nasopharyngeal tumors should be treated. Allergies should be treated with antihistamines and, where indicated, by desensitization.

If the fluid does not clear with medical treatment within a week or two, a myringotomy should be done. If there is a question of active infection or if the fluid looks purulent, as is seen at the conclusion of acute otitis, cultures are taken. On adults this can be done in the office without anesthesia. It is no more painful than an intravenous needle for a blood test. A good safe topical anesthetic has a tremendous psychological value to the patient. Children under the age of 1 require no anesthesia. Between the ages of 1 and 3 anesthesia is not absolutely essential although a general anesthetic may be used to avoid the child's possible mistrust at follow-up examinations. I usually do the myringotomy at the same time as the adenoidectomy if the adenoids are enlarged. Once drainage has been established with decongestants or by myringotomy, positive pressure inflation of the middle ear is invaluable in forcing out the serous fluid and keeping it from reforming. The patient can do this himself by performing the Valsalva maneuver. This should be done several times a day, by taking a deep breath, holding the nose and blowing hard against the closed lips for a second or two.

In resistant cases where the fluid reforms as soon as the myringotomy heals, small polyethylene tubes are inserted through the myringotomy site. Insertion of the tubes, even in adults, usually requires an anesthetic. Good anesthesia can be obtained by infiltrating the canal wall with a local anesthetic, using a #27 needle. Once inserted the patient has no sensation of the tubes' presence. Be careful to caution the patient or parents not to allow any water to get into the ear canal while a myringotomy is open or a tube is in place. Water can be kept out by a pledget of lamb's wool in the ear canal or a cotton pledget thickly coated on the outside with petroleum jelly. I check these patients a week after a myringotomy to be sure it is healed and at two month intervals until the tubes have fallen out, usually within three to six months after insertion.

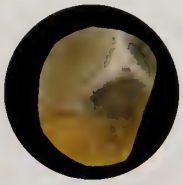
The mucoid type of fluid is so thick and tenacious that it is appropriately called a glue ear. It is aspirated only with difficulty through the eardrum, and occasionally must be removed through a tympanotomy. This thick, glue-like fluid is prone to recur, as the myringotomy usually heals long before the mucous membrane has returned to normal. Repeated myringotomies, as many as ten or twenty, were formerly required for this condition. Now polyethylene tubes are inserted initially when this thick, glue-like material is found. At times a subacute mastoiditis may accompany the serous otitis, which will necessitate a simple mastoidectomy before the condition can be eradicated. Usually the thin serous fluid readily responds to decongestants or to a myringotomy and removal of the eustachian tube obstruction. Occasionally, however, even serous fluid will repeatedly reform. For this, resection of the tympanic plexus of nerves which lies on the promontory of the middle ear has been carried out, as the tympanic branch of the glossopharyngeal nerve is the secretomotor nerve to the ear.

Failure to diagnose serous otitis is the most common cause of the recurrent, almost continuous otitis me-

Otoscopic Views



Normal



Acute Serous



Acute Purulent



Chronic Serous

dia seen in young children. In these cases a myringotomy will frequently provide a long-term cure without adenoidectomy or any other surgery. Failure to do this may allow the condition to go on to acute or subacute mastoiditis. Adhesive otitis media with scar tissue binding down the ossicles or tympanosclerosis may also result with permanent impairment of hearing. The prognosis for hearing with adhesive otitis or tympanosclerosis is usually poor. If the adhesions are removed, they reform, and the same is frequently true of tympanosclerosis even after a tympanoplasty. For many of these people a hearing aid is the only solution. The constant negative pressure in long standing serous otitis media may draw in either Schrapnell's membrane or the posterior superior portion of the eardrum forming a pocket to cause a chronic otitis media with cholesteatoma, which may not become apparent until twenty or thirty years later.

The non-medical complications of improper diagnosis or treatment may be even more serious. The irritable child, the frequent bouts of acute purulent otitis media with pain and fever, the expense of antibiotics and doctors, and the time lost from school or work affect the entire family.

In summary, serous otitis has many causes. There is a blocking of the eustachian tube and an inflammatory reaction in the middle ear mucosa, the latter usually caused by a mild infection. The treatment is to provide immediate drainage by decongestants and where necessary, by a myringotomy. The obstructing agent must be removed by surgery if it is adenoid or by oral decongestants, and antihistamines or nasal spray if it is an upper respiratory infection or allergy. Occasionally resistant cases require plastic tubes placed through the eardrum or other more radical surgery. Failure to treat properly leads to hearing loss which may be permanent, repeated acute otitis media or possibly even chronic otitis media with cholesteatoma.

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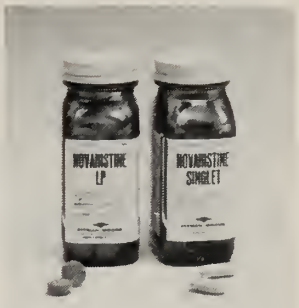
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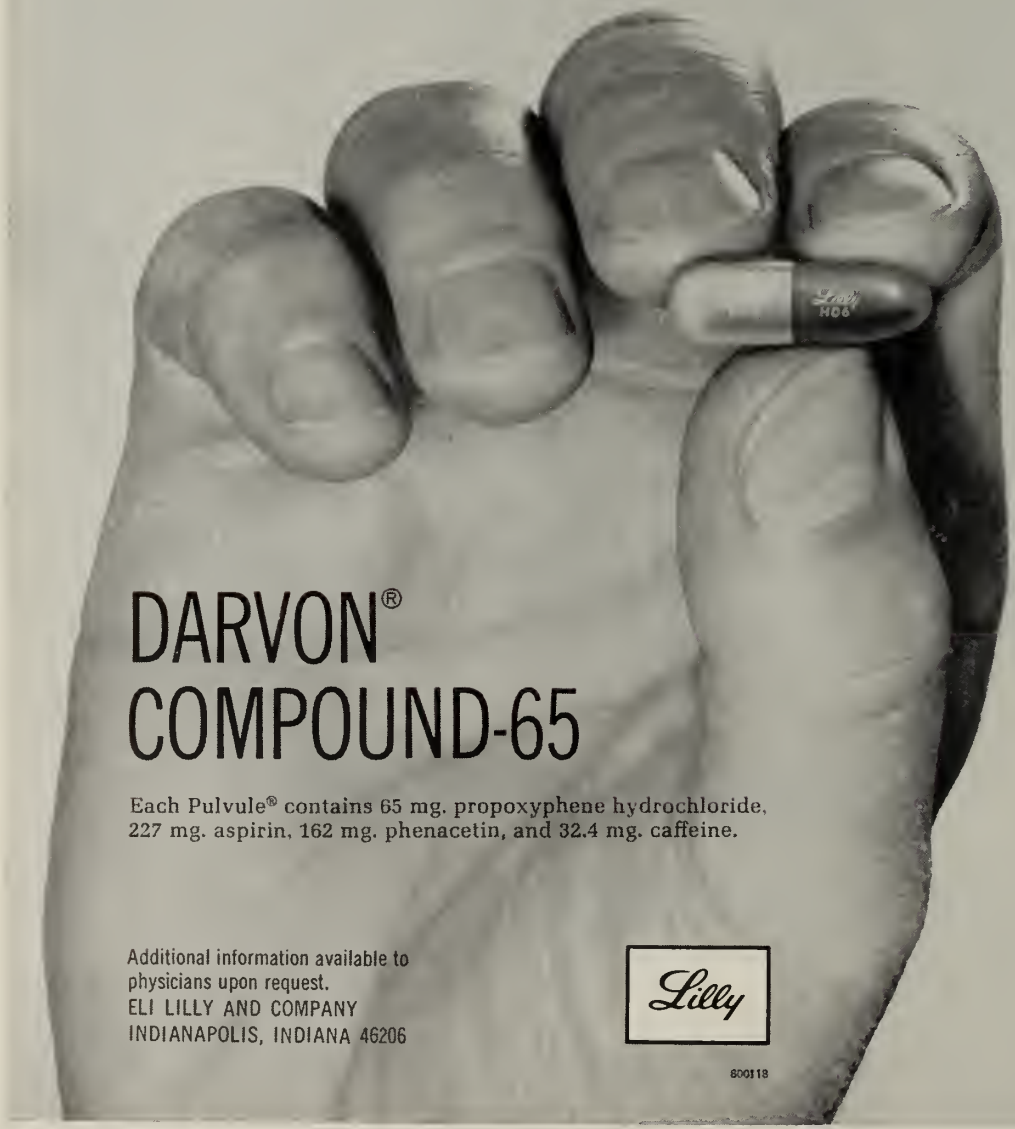
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Guest Editorial....

Incompatibilities—*In Vitro* and *In Vivo*

INCOMPATIBLE is probably best known as the word frequently used to describe two people of a union that had failed as a marriage. And in that sense it is used to define what is almost indefinable. But in the study of pharmacy, incompatibilities is a fascinating subject concerned with drugs and their combinations when they fail to "marry" in union with one another. The fact that certain powders, when rubbed together, will liquefy means that they are incompatible and so also are certain liquids which cause an undesirable precipitate when combined in the same mixture. The knowledge of these facts and others, similarly related, has stood the pharmacist in good stead for many years and has enabled him to compound medication of pharmaceutical elegance. Now, however, such information is seldom needed in prescription compounding due to the widespread use of ready-made preparations.

But with the decline in the importance of incompatibility *in vitro*, there is another, possibly more pressing, aspect developing—incompatibility *in vivo*. This drug action, or reaction, has drawn more attention since the occurrence of what might be termed the "armamentarium explosion", meaning the discoveries of the "wonder drugs" and all the related products and advancements which they engendered.

As the result of these so-called incompatibilities *in vivo* we find warnings appearing in the literature with certain drugs. Not uncommon is the statement that "patient should be cautioned against driving a car or operating machinery", or "patient's tolerance to alcohol may be lowered", or "the response to alcohol may be potentiated", or "avoid unnecessary exposure to sunlight". Although devoid of the pharmacist's responsibility and certainly not in his immediate sphere of practice, the thought occurs

that cooperation resulting from a meeting of minds between physicians and pharmacists could initiate positive steps that would be helpful to the patient. For example, today, the pharmacist sometimes attaches auxiliary labels to prescriptions, such as "Shake Well Before Using" or "For External Use Only", so that the medication will be properly used. Now, taking into consideration the aforementioned precautions concerning certain drugs and assuming that agreement has been reached at the top levels of medicine and pharmacy, there could be additional auxiliary labels which the pharmacist would use with certain prescriptions. For example, such labels might read "Don't drive an automobile or operate machinery", or "Alcohol should be avoided", or "Avoid excessive sunlight". More extensive and thorough study of the subject might well indicate additional "labels", or if not more auxiliary labels, possibly pertinent advice, when indicated, could be typewritten on the prescription label.

Such a program would naturally be instituted by the medical profession and although it would require much technical as well as practical groundwork, the efforts might be well worthwhile.

The rendering of this service to the patient would add to the pharmacist's *raison d'etre* and might possibly relieve the work load of the physician.

William G. Tarrant, Jr.

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1 West Broad Street
Richmond, Virginia 23220

Light Coagulation

DuPONT GUERRY, III, M.D.
Richmond, Virginia

Light coagulation has become an important modality in the treatment of many ocular disorders that have been difficult or impossible to treat in the past. Of the two instruments available, the xenon arc and the ruby laser, the former is considered best suited for carrying out routine light coagulation. It is widely used in the prophylactic treatment of retinal detachment.

TEN YEARS AGO the first case of clinical light coagulation carried out in this country was reported by our group[1] before the Virginia Society of Ophthalmology and Otolaryngology. It would, therefore, seem appropriate a decade later to review the problem of light coagulation, to indulge in a sort of "light coagulation revisited", as it were.

The precursor of clinical light coagulation was the retinal burn resulting from exposure to sunlight, usually during an eclipse. Historically it is known that the ancients were well aware of visual loss resulting from such exposure as is seen, for example, in Plato's dialogue, the Phaedo. In this dialogue Socrates states "I thought that as I had failed in the contemplation of true existence, I ought to be careful that I did not lose the eye of my soul; as people may injure their bodily eye by observing and gazing on the sun during an eclipse unless they take the precaution of only looking at the image reflected in the water or in some similar medium." In the second century A. D., Galen described eclipse blindness and during the renaissance Gallileo, while making observations on the sun with his telescope, received a macular burn. The first clinical description of an eclipse burn was made by Bonita and the first ophthalmoscopic appearance of an eclipse burn was reported by Cocceius[2] in 1853. Since that time a considerable number of macular burns have been reported, the largest series consisting of some 200 cases which were observed by Birch-Hirschfeld[3] and Blessig[4] in 1912 following the eclipse of that year. In 1944 Cordes,[5] during World War II, reported macular burns in military personnel who inadvertently followed Japanese planes into the sun with their binoculars.

Clinical reports of visual loss following exposure to sunlight triggered numerous experimental studies. The most important of these was that by Verhoff and Bell[6] in 1916. These investigators demonstrated that eclipse burns and burns from the arc lamp as well were thermal lesions caused by the transformation of light energy, impinging on the pigment epithelium, to heat. As nuclear weapons developed it became apparent that atomic and hydrogen weapons were capable of producing retinal damage at distances where the inverse square loss was no longer operative. Buettner and Rose were the first to warn of this hazard.

With the knowledge of retinal burns both from eclipse exposures as well as exposures to the carbon arc lamp at hand, Meyer-

President's Address given before the Virginia Society of Ophthalmology and Otolaryngology at Hot Springs, April 27, 1967.

Schwickerath[7] realized the clinical implications and set about developing a practical method of carrying out clinical light coagulation. He first employed sunlight as a light source. This was followed later by a carbon arc, and, finally, in collaboration with Littman, the definitive Zeiss light coagulator with its xenon arc came into being.

Independently, while Meyer-Schwickerrath was carrying on his clinical experiments, Guerry, Ham and their co-workers in the Departments of Ophthalmology and Biophysics at the Medical College of Virginia began their collaborative study on the thermal effects of visible light on the retina. It was these studies, carried out for the purpose of determining the threshold dose which would lead to irreversible damage to the retina that led us to the realization that experimental light coagulation had clinical overtones. In 1957, as already mentioned, the first case of clinical light coagulation carried out in this country was burned in our laboratories with the experimental instrument. This case was a 20 year old white male with a central von Hippel's hemangioma, 2 to 3 disc diameters in size, in the right eye. The patient looked directly into the beam of the carbon arc searchlight which served as light source and light coagulation was carried out by triggering a rotating shutter. This treatment resulted in complete obliteration of the tumor. The patient now (ten years later) has a visual acuity of 20/60 with only a moderate paracentral eschar and scotoma. The patient's acuity prior to light coagulation had been a poor 20/200 with a dense central field defect.

When the Zeiss light coagulator became available to us in 1958, we began to use it both clinically and experimentally. In the nine years which have elapsed we have treated over 4,000 human eyes. Five years ago a research coagulator was constructed along lines similar to the conventional coagulator but with different optics and with a xenon arc source capable of being pulsed. Since that time this research coagulator has been used in most of our experiments, the conventional coagulator being employed entirely as a clinical tool.

As might have been predicted, the discovery and development of the laser principle by Townes, followed by Maiman's demonstration of the ruby solid state laser, led to the use of the laser as a light source for photo-coagulation. And today a myriad of lasers such as the solid state laser, liquid lasers, gaseous lasers, and the ingenious Q-switched laser, which allows one to work in the almost unbelievably short nonasecond range, are available. The power ranges of these devices varies from milli to giga watts and they may be pulsed or may be used to produce continuous radiation. Characteristically the unique properties of the laser beam is in its directionality, monochromaticity, coherence and polarization. Undoubtedly lasers can be developed for almost any spectral range and for almost any purpose.

When light coagulation was first used clinically it at once became apparent that its greatest use would be in the prophylactic treatment of retinal detachments. Since the work of Gonin, retinal tears were recognized as the cause of the great majority of retinal detachments. It was logical, therefore, to assume that if such tears could be sealed off, detachments would not develop. Prophylactic treatment of retinal tears had been suggested prior to the advent of light coagulation, but surgical intervention was then necessary. Most surgeons and, indeed, most patients refused prophylactic treatment and only underwent surgery when detachment occurred. The simplicity and bloodlessness of light coagulation immediately recommended this mode of treatment to surgeon and patient alike and the original prediction that this would be perhaps the light coagulator's most important contribution has been borne out by the passage of time.

All fresh tears of the retina should be

coagulated immediately.

Most retinal tears make themselves known by such entopic phenomenon as "spots in front of the eyes" or "light flashes". The so-called "benign senile light flash" which manifests itself as a generalized crescentic shaped peripherally located phenomenon is of no significance and probably is never associated with a true retinal tear. The point located, flickering type of light flash, however, is clinically significant and many of these are associated with a true tear or with a vitreo-retinal adhesion which is potentially capable of tearing. The spots which are associated with retinal tears are apparently derived from two sources: 1. capillary or larger vessel hemorrhage, or 2. particulate matter derived from retina and incorporated in the vitreous where it detaches from the retina. When either or both of these phenomena are complained of, the patient should be examined immediately and thoroughly with the pupil maximally dilated. If the vitreous is too muddy from hemorrhage for accurate retinal appraisal, the patient should be put on absolute bedrest with stenopeic specs until such time as the vitreous clears and the fundus can be evaluated.

In cases of peripheral holes which are nearly always of the degenerative type there is usually a tiny modicum of pigment surround. This will usually prevent detachment. If, however, it can be clinically established that such a hole has not spontaneously sealed itself, then prophylactic light coagulation is indicated.

Perhaps the commonest place to find a retinal tear is the upper temporal quadrant just posterior to the equator over the insertion of the superior oblique muscle. Numerous authors have shown statistically that the greatest percentage of retinal tears occur in this region and it is surprising how often retinal pathology will be found when this area is examined. Furthermore, when an actual tear occurs in this area in one eye, the fellow eye may exhibit changes of similar character which are precursors of a retinal tear. Everett has used the term "the fellow eye syndrome", to emphasize the frequency of mirror image pathology. Where such pathology occurs, bilateral prophylactic therapy is indicated.

Another type of hole, where there may be few prodromal symptoms, are the holes seen in lattice degeneration of the retina. The underlying pathology in lattice degeneration has not yet been fully elucidated, but, from a pragmatic standpoint, whenever this condition occurs retinal detachment may result. This is due to the development of through and through degenerative holes in the areas of lattice involvement. It is our feeling that all of the lattice areas should not be prophylactically treated, but only those areas where definite holes develop. Constant surveillance of these patients is necessary and they should be examined at least twice a year with pupils widely dilated; all of the lattice areas should be thoroughly inspected and if new or potential holes have developed, these should be treated.

Unquestionably holes located above the horizontal meridian carry a greater detachment hazard then do those located below the horizontal meridian. This is due to the fact that gravity has an adverse effect where the lesion is above but is of no consequence where the lesion is below. When light coagulation is carried out a much firmer barrage should be laid down around holes above the horizontal meridian than around those situated below. In addition, post light coagulation, the patient should be immobilized much longer.

In holes occurring at the extreme periphery just at the ora or pars plana light coagulation may be difficult to carry out. By indentation and with maximal dilatation of the pupil, however, any hole can be light coagulated. In the past we have found it occasionally expedient to use the external coagulator transsclerally in treating such peripheral holes. Today, now that cryotherapy is available, in many instances it will be found simpler to treat the peripheral

hole through conjunctiva with the cryoprobe rather than to use light coagulation. Here one's clinical acumen must come into play.

Light coagulation is the treatment of choice for macular holes. The difficulty here, however, is not in deciding on what is the best method of treating such a hole, but rather what is and is not a hole. The occurrence of a true through and through macular hole is extremely rare and unless detachment actually takes place, the only certain method of determining the presence or absence of a true hole is by diagnostic light coagulation. It has been said that whenever an operculum occurs overlying a macular lesion, a hole unequivocally exists. This statement is misleading as I have seen several cases in the past five years with opercula overlying a macular lesion suggestive of a hole and yet the lesions were cystic in character. These were light coagulated and a definite coagulum formed at the depths of the crater indicating that the lesion did not involve the outer layers. The obvious cases of macular cysts almost without exception are dotted with yellowish spots. These spots apparently are remnants of Mueller's fibers and when they occur, one can be positive that a true hole does not exist. Such macular cysts do not need light coagulating.

There are cases where one cannot decide whether or not a true hole exists and it is our feeling that in such instances if the patient's vision is 20/200 or less, prophylactic light coagulation should be carried out. This will prevent detachment if there is a true hole and will not lead to any further loss of vision if the lesion proves to be cystic in character.

In most cases of true retinal detachment light coagulation is of little value. Occasionally, however, a minimal peripheral detachment can be easily barraged off, the barrage tying into the ora at either end of the detachment. Furthermore, a small number of flat detachments will almost completely disappear on bedrest to the extent that the offending hole can be light coagulated. A fair number of these immediately recur on ambulation and, if such a case is light coagulated, ambulation should be slow and stenopeic specs worn for a considerable period of time.

In the management of a case of true retinal detachment, light coagulation does have a very important role to play. Many times the surgeon is somewhat nonplussed to find post-operatively that there is not enough reaction present to close off a given hole although the retina may be very well co-apted to the underlying pigment epithelium on top of a plomben or buckle. Such cases lend themselves nicely to light coagulation and re-operation is not usually necessary.

At a later date in the post-operative period, leaks over a buckle may be noted. These can in most instances be walled off and dried up by light coagulation of the offending area.

The management of retinoschisis has been somewhat controversial. It is our feeling that early cases of retinoschisis, where the splitting process is confined to the periphery, should be left alone and simply observed at intervals to be certain that holes do not develop in either the outer or inner lamella of the schised area. If the splitting process is progressive, then light coagulation should be carried out and the area of schisis completely isolated by a double barrage, one line of which should straddle the demarcation line of schisis and the other placed contiguous to this in sound retina. If holes develop in either the inner or outer lamella, this is also cause for isolating the schisis with a barrage. Furthermore, if a case of retinoschisis has advanced as far as the equator, light coagulation should also be employed to prevent its spread. In the advanced cases where the macula is threatened it is imperative that light coagulation be carried out immediately and the schised area walled off completely with a double barrage.

We have had two cases of this sort which have split to within a disc diameter or two of the macula and following light coagulation there has been no progress.

Vascular lesions of the retina lend themselves beautifully to light coagulation. Most of these lesions, although they are not malignant in the usual sense, cause loss of sight either by hemorrhaging or by destruction of the retina by growth or detachment.

Von Hippel's hemangiomata of the retina can in many instances be completely obliterated by light coagulation. The small saccular hemangioma needs only a few exposures of mild intensity to carry out complete obliteration. The larger hemangioma, involving as much as an eighth to a quadrant of the retina, is difficult to obliterate by light. Combined therapy, diathermy and light coagulation must be employed. The reason for this is the fact that light does not penetrate well into the depths of the tumor and many of the hemangiomas, as a result of fibrosis, are somewhat light colored and tend to reflect and not absorb light. Combined therapy, diathermy and light coagulation, is effective in lesions involving up to about a quarter of the retina. In lesions involving an area greater than this, diathermy and light coagulation intense enough to obliterate the tumor usually results in loss of sight from vitreous shrinkage followed by phthisis.

Retinal teleangiectasis which occurs very rarely may lead to loss of sight, either by hemorrhage into the vitreous or by developing into a definitive Coat's disease. This latter process is indeed rare, but it does occur. In cases of retinal teleangiectasis the teleangiectatic areas should be completely obliterated. If this is done in the early stages, only a small amount of retina is sacrificed and there is no loss of vision.

If teleangiectasis has developed into Coat's disease or if early Coat's disease is discovered in a retina, there have been no recognizable teleangiectatic precursor, the Coat's tumorous mass can be obliterated by light coagulation. Light coagulation alone should not be used in Coat's disease involving more than an eighth of the retina and if the lesion is larger than that, combined diathermy and light coagulation should be carried out. In the massive case of Coat's disease which is the type usually seen, we have had no luck with any form of therapy and most of these eyes eventually develop complete detachment followed by phthisis and enucleation.

Eale's disease has been one of the most difficult ophthalmic diseases to treat and in the past a high percentage of sufferers from this disease have lost all useful sight. Recurrent hemorrhages followed by organization frequently result in traction detachment with eventual phthisis. Until the advent of the light coagulator there was no satisfactory method of treating these cases. A great majority can now be adequately treated and useful vision salvaged, if therapy is instituted early enough. The areas of neovascularization should be lightly treated with the light coagulator and as new vessels crop up they should be obliterated before hemorrhage takes place. Ultimately the disease becomes stabilized and there is no further progress. In most cases the area of the macula is spared although not infrequently areas very close to the macula must be treated. It is extraordinary, however, how little visual impairment results from these treatment areas although negative scotomata can at times be plotted where treatment has been carried out.

Diabetic retinopathy today is one of the principal causes of blindness. No real satisfactory form of therapy exists. Ablation of the pituitary is helpful in controlling some cases, but this procedure, even though the morbidity of the surgical procedure is low, leaves the patient a pituitaryless animal dependent upon substitutive steroid therapy for maintenance of life itself.

Recently, light coagulation of diabetic retinopathy has been popularized. While the retinopathy cannot be cured, in many instances it may be controlled. Light coag-

ulation in severe diabetic retinopathy with advanced traction detachment, following repeated hemorrhages from proliferans, is of no value. Where retinopathy is confined to superficial vascularization with little proliferans extending into the vitreous, in other words cases resembling Eale's disease, light coagulation is most effective. We have a collaborative study with the Department of Neurosurgery at the Medical College of Virginia where a comparative study of pituitary ablation and retinal light coagulation is being undertaken. Although the cases are still too few in number for anything but a clinical impression it appears that light coagulation is more effective than pituitary ablation.

The treatment of active lesions of chorioretinitis leaves much to be desired. In most cases, particularly of large isolated chorioretinal foci, the disease is self-limited. When healing has once occurred there is an end of the process. And unless the vital macular area has been destroyed, there may be little or no visual loss once the vitreous has cleared. We have light coagulated many active foci and a sufficiently high percentage show more rapid improvement than is seen in cases treated by conventional medical means, to make such therapy worthwhile. Our plan of attack has been to coagulate the entire periphery of the chorioretinal lesion with moderately intense exposures.

Of more dramatic import, however, is prophylactic treatment of recurrent attacks of chorioretinitis, particularly of that type clinically diagnosed as toxoplasmic retinochoroiditis. Some years ago we reported the prevention of recurrent attacks of central toxoplasmic retinochoroiditis by coagulating the affected area where central vision had already been destroyed. In these few cases no recurrence has occurred. Spalter[8] and his co-workers have recently reported treating paramacular foci of retinochoroiditis in an effort to prevent re-activation of previous lesions or development of new lesions contiguous to the macula. They have employed the AO laser with gratifying results. Such therapy as close to the vital macular area as 1/2 to 1/4 of a disc diameter leaves no room for miscalculation. The speed of delivery of light energy with the laser would seem to make this instrument ideal for such therapy.

Another recent innovation where light coagulation has proved to be of value is in the treatment of cases of central angiospastic retinopathy. In employing such therapy, the leaking choroidal area is first identified by intravenous fluorescein injection and then the offending spot is light coagulated. This can be done with the conventional light coagulator or the laser. Following such therapy, edema disappears rapidly, usually within a few days, and acuity returns. This type therapy is of no value in the central edema seen following cataract extraction, where pathology is apparently retinal and not choroidal.

Light coagulation in the treatment of malignant tumors of the retina was employed very early. First reports concerning the treatment of malignant melanoma in the adult and retinoblastoma in the child were most encouraging. Follow-up data which has accrued in recent years indicates, however, that while some cures have been effected there have been many untoward results. Where the tumor has not been completely obliterated a false sense of security has resulted in late enucleation followed by metastatic spread. Consensus has it that such treatment should be reserved for only eyes and not used routinely. In the treatment of retinoblastoma light coagulation serves a useful purpose when used in conjunction with chemotherapy and radiation.

Malignant melanoma of the iris is apparently an entirely different type of lesion from the standpoint of both local and metastatic spread. Malignant melanoma of the iris can easily be excised by a complete root iridectomy unless there is invasion of the ciliary body and angle, but even these

cases are not hopeless. We have had a case in the last five years where there was moderate invasion of the angle, but not of the ciliary body. Here a root iridectomy was carried out and yet angle involvement was still present. Light coagulation of the angle with the external coagulator and with a Koeppe type gonioscopic lens in place has apparently resulted in complete eradication of the tumor cells remaining in the angle.

The external light coagulator is valuable in the treatment of several disorders, not only of the anterior segment, but of the adnexa as well. The creation of a new pupil or the enlargement of an old pupil in an aphakic eye is easily effected with the light coagulator. The coagulator is set at a high intensity level in the red zone and the beam is carefully focussed on the area where a new pupil is to be created. As energy is delivered a popping noise is heard and at the same time the operator sees a new pupil blown open by an instantaneous explosion. If there is no secondary membrane behind the new pupil, visual improvement will be noted immediately.

This procedure is most valuable in cases of hammock or updrawn pupil and in cases where the upper half of the cornea is clouded by corneal edema which covers the pupillary aperture.

The obliteration of epithelial cysts in the anterior chamber can be effectively carried out in most instances by light coagulation. In such instances the beam should be focussed on that part of the cyst which is in contact with the light absorbing iris tissue. As energy is delivered the iris tissue is seen to shrink and the contents of the epithelial cyst turn somewhat milky white in color. As a rule, two or three sessions of light coagulation are necessary to obliterate such a cyst unless the cyst, when first recognized, is extremely small in size.

Small hemangiomas of the lid or conjunctiva, papillomas and in particular xanthelasmic patches can be nicely obliterated by light coagulation. Most such lesions can be adequately treated by other methods and in such instances, therefore, light coagulation is more exotic than pragmatic.

Complications of light coagulation: The most dreaded complication in light coagulation is that of vitreous shrinkage. This comes about as a result of employing too high intensity and if the treated area is large enough and intensity strong enough a complete traction detachment of the retina may result. Such a complication never occurs if therapeutic doses are employed and if repetitive treatment is carried out over a period of time rather than attempting to treat too large an area at one session.

Another complication is that of hemorrhage into the vitreous. Here again, this complication is not seen unless very large doses outside the therapeutic range are used, and a retinal vessel inadvertently treated. The affected vessel may simply explode or later necrosis may take place. In any event, massive hemorrhage into the vitreous occurs.

Another complication of serious import is the accidental burning of the fovea. This can occur when parafoveal chorioretinal lesions are being treated, either prophylactically or in an effort to cut down the activity of chorioretinitis. There is a very real danger in treating such lesions as the patient may suddenly fixate the light source at the exact instant the coagulator is triggered. The best method of minimizing such a catastrophe is to carry out light coagulation under general anesthesia with the eye completely immobile.

Mild iridocyclitis may occur following light coagulation, particularly if the pupil is not widely dilated, and the iris is allowed to absorb an inordinate amount of energy. Concomitant with iridocyclitis, a severe secondary glaucoma may result where even more energy is absorbed than that which gives rise to a mild iridocyclitis.

Irreversible damage to the cornea has also been reported following intensive light coagulation at high energy levels. Such corneal damage may be irreversible. A mild keratitis

is not uncommon following prolonged light coagulation, but this is of no significance and clears within a few hours under local steroid therapy.

Corneal burns following the use of the anterior segment coagulator, or the external coagulator, are not uncommon and, unless very severe, disappear after a period of months without sequelae.

From what has been said, it is apparent that light coagulation is an important modality in the treatment of many ocular disorders that have been difficult or impossible to treat in the past. In carrying out light coagulation the clinical ophthalmologist has at his disposal two principal types of coagulators, the xenon arc and the ruby laser. We feel that the versatility, wide spectral range, safety features, ease of operation and visual control all make the Zeiss light coagulator the instrument of choice for carrying out routine light coagulation and would reserve the laser for cases where energy must be delivered rapidly as in the prophylactic treatment of paracentral retinochoroiditis.

What of the future of light coagulation? It is probable that other light sources and simpler or more convenient methods of delivering light energy will be developed. Research in this direction, in the field of eye protection from high intensity light, and in the field of weaponry is being avidly pursued.

Thus, we see the eclipse burn of the ancients now a practical clinical tool, no longer a destroyer of sight, but rather harnessed and directed into healing channels for the benefit of mankind.

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Reconstructive Procedures in Head and Neck Cancer Surgery

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Cancers of the head and neck are best treated by surgical removal. Sometimes because of the structure involved, the location, and the size of the tumor, reconstruction becomes a problem of major importance. The technique for dealing with these cancers is presented.

DESPITE THE REFINEMENTS in radiation therapy which have become manifest during the past two decades, surgical excision still remains the most effective method of treating cancers originating in the extracranial supraclavicular portion of the body. The integument of the head and neck is the site of the vast preponderance of such tumors but the mucous membrane-lined recesses of the cranium, oral cavity and pharynx harbor cancers which are more difficult to locate and whose treatment produces vast changes in structure and function. Such tumors are generally epidermoid carcinoma or one of its variants. They often grow to great size and may involve multiple vital structures before they are diagnosed and referred for definitive treatment. Despite the accessibility of such tumors, the sad fact remains that inadequate examination, procrastination and ignorance are the chief reasons for failure in the treatment of such tumors. Often the patient has been previously referred to the radiotherapist for what was hoped to be definitive therapy and thereby eliminate the need for surgery. Too often such treatment is anything but palliative (Fig. 1). As a last resort, in a confused and complex medical situation, the patient is referred to the surgeon. The surgeon faced with such cases is well aware of the hazards of his techniques. First of all, he must delineate a neoplastic process whose borders have been obscured by the tissue changes associated with radiation treatment (Fig. 2). In such cases, the planned resection must include all suspicious tissues and therefore the magnitude of such procedures is great. He also realizes the vast array of wound complications which may develop in such patients. Lastly, the surgeon is faced with wound restitution, without the usual benefits afforded by time. Reconstruction is still the major issue with



Fig. 1. Radiation ulcer with exposed mandible, marked trismus, cancer invading cheek and neck. Previously treated with pull-through resection and 10,000r.

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which the operator is faced (Figs. 3 and 4). All of the steps employed in the ablation of the primary cancer must be successfully integrated with a well conceived blueprint of the proposed reconstruction. If the surgeon has approached the tumor with proper respect for management of the resultant



Fig. 2. Resection included cheek, neck, jaw, and tongue with common and internal carotid arteries.



Fig. 3. Local neck flap rotated into upper neck defect and stage reconstruction with cross chest flap.

3-dimensional defect, and if he has protected local tissues from haphazard and poorly placed incisions, the reconstruction of the wound is often a relatively simple matter. On the other hand, should the neck be opened through incisions which have been made by rote rather than reason, reconstruction may be obviated as a feasible



Fig. 4. Patients free of disease over five years following treatment. Chest flap lines, oral cavity and pharynx.

procedure. Even more fallacious is the reasoning that adequate tumor removal alone should suffice and no respectable planned reconstruction is either desirable or necessary. Unfortunately, the latter opinion has prevailed in some of the country's leading medical centers until recent years.

The successful treatment of cancer of the head and neck demands a sound knowledge of plastic and reconstructive surgery. Such knowledge must be based on the experience gained from planning the entire procedure. Anatomical barriers encountered in the extirpative process must be entered and destroyed in developing the specimen. A panoramic picture of the operative defect should be indelibly etched in the surgeon's mind long before the composite specimen is handed from the table. The possible need for transfer of local or distal flaps should

also be vivid in the mind's eye of the operator. As the moment of truth approaches, the informed surgeon is irrevocably impressed with the basic tenet of the situation: the removal of the tumor must include a clear and concise picture of reconstruction. The scope of the subject is so vast that I will limit my discussion to those cancers which originate in the upper alimentary tract.

The surgical treatment of cancer of the lip will be discussed in more detail by another essayist, so my remarks will be brief. These cancers, all epidermoid in nature, constitute the largest group of cancers of the upper alimentary tract. They metastasize to the lymph nodes of the neck in about 14% of patients. They can be cured by adequate removal of the primary tumor with or without the pathways of lymphatic spread depending on the stage of the tumor.



Fig. 5. Resection of entire lower lip for epidermoid carcinoma. Gillies' flap rotated into defect and donor site closed primarily.

Resections which include no more than 30% of the lower lip can be closed primarily without producing any defects in appearance or function. Larger defects require pedicle flap closure (Figs. 5 and 6). The Estlander flap, modified by Sir Harod Gillies, has proved to be the most effective method of reconstruction and often the entire lip can be reconstructed in two operations.[1]



Fig. 6. Patient one year after resection. Both commissures opened under local one month after first operation.

Advancement flaps have been used to a lesser degree, and although the operative defect can be filled without difficulty, it produces a flat, wide mouth often with none of the natural features of the Estlander reconstruction.

Cancers of the gingiva, floor of mouth, and tongue differ markedly from the standpoint of prognosis. The surgical treatment, however, produces similar defects which often tax the imagination of the operating surgeon. Suffice it to say, any extirpative procedure which eliminates one-half of the floor of mouth, significant segments of the tongue root, and pharynx will require replacement of the resected lining. Many surgeons in this field have advocated the use of free skin grafts fixed in place with some form of stent. Experience with this method of wound management has been

discouraging. Particularly has it been so in those cases which have been previously treated with definitive amounts of radiation. Stent extrusion with wound breakdown has been encountered in a significant number of cases. Carotid disruption with fatal hemorrhage has occurred early in the postoperative course.[2] Varying degrees of contracture have occurred in all of this group of patients. The use of pedicle flaps has been the treatment of choice in this group of patients. Initially, we elected to create a surgical pharyngostoma in all such patients. Through this avenue, a pedicle flap raised on the neck, chest or shoulder was introduced as the intermediate step in the reconstruction (Figs. 7 and 8). This group



Fig. 7. Transthoracic pedicle flap secondarily introduced through neck incision to replace resected tongue and floor of mouth.

of cases has enjoyed almost uniform success and serious wound complications have not occurred. Its major objection is related to the flow of saliva on the neck, into the tracheostoma and dressings. Another real objection is related to the time involved and the number of procedures necessary for successful reconstruction. Two years ago, I became interested in the use of the forehead as the source of lining. Although we have used the scalp as coverage for over ten years,[3] it was only recently with Mil-



Fig. 8. Patient nine years after subtotal glossectomy with floor of mouth and neck dissection for mucoepidermoid carcinoma of tongue. Pedicle flap restores floor of mouth.

lard's work that the frontal scalp has been used in replacing lining material in the floor of mouth and pharynx.[4] This flap has more versatility than any local tissue utilized in reconstructing major oral cavity losses. The only real objection to its use is that of altering the facial countenance by eliminating the natural forehead wrinkles with a split graft. The elevation of the flap adds significant time to an already lengthy operation. Millard has recommended the operation be divided into two parts. The phase of wound restitution is completed by an entirely new team of surgeons. Theoretically, this should eliminate the laborious task of tumor removal and reduce the rigors of such an undertaking. Its obvious deterrent is that of divided responsibility. In other regional surgery, divided responsibility has generally led to inferior and inconclusive results. The spectacular results are enjoyed in communal fashion, but the failures never seem to be the responsibility of any individual.

In a previous publication, the plea for extending the resections in advanced cancers has been documented.[2] Of this group,

the recurrent tumors in the laryngopharynx have all required the use of pedicle flaps raised on the chest, and local flaps raised on the neck for lining. Obviously the plan for neck incisions in this group must be given priority attention. The salvage rate in this group has been most rewarding. Except for two cases, all patients have been so reconstructed that alimentation has not been a serious problem upon completion. Often such a reconstructive endeavor can be completed in ninety days (Fig. 9). Others have required as long as one year (Fig. 10). The most significant contribution to this field of head and neck surgery was made by Bakamjian of Roswell Park.[5] He has designed a flap which eliminates the multitude of intermediate steps heretofore described. In two operations, or about six weeks after its initiation, the swallowing process has been reconstituted. We have found this flap to be useful in controlled resections of the face and maxillofacial skeleton (Fig. 11) but, to date, have not employed it in reconstruction of the pharynx and cervical esophagus.



Fig. 9. Patient three years after resection of carcinoma of hypopharynx with cervical lymph node metastases. Swallows normal diet without difficulty.

Discussion

It should be apparent from this presentation that the treatment of cancer of the head and neck belongs to no specialty of medicine. Such surgery is best handled by those who have been well-grounded in the rudiments of pathology, anatomy, and general surgery. Proper training in postoperative management of patients who are often critically ill is also an obvious addition to the list of prerequisites. The one unalterable fact is that this form of surgery generally fails because of the wound created by monobloc removal of neoplastic tissue. It is still primarily a surgery of wound management. No amount of knowledge related to blood gases and fluid shifts or even more sophisticated information can solve the dilemma of reconstituting major losses of the floor of mouth, tongue and pharynx. This critical information belongs only to the



Fig. 10. Patient nearly three years after resection of pharyngoesophagus with bilateral radical neck dissections. Swallows soft foods well.

surgeon who is thoroughly familiar with the principles of plastic and reconstructive surgery. Today the magnitude of operations for those cancers arising in the head and neck has been successfully extended to the maximum by those surgeons, who, by

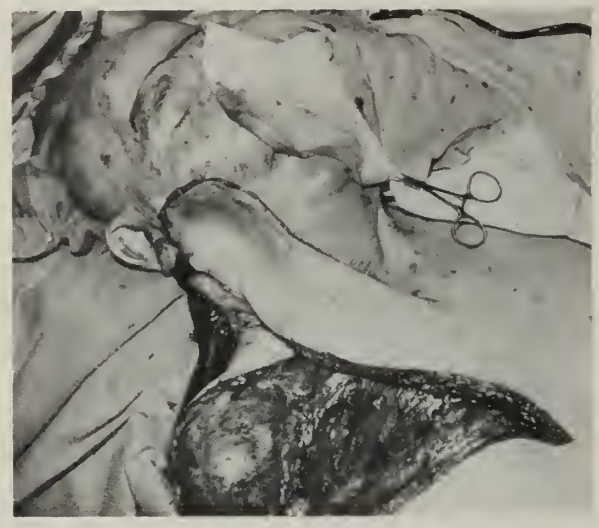


Fig. 11. Wound restitution with parasternal-based cross chest flap. Resection for advanced carcinoma of parotid in 88 year-old male.

virtue of their background, have no fear or concern for the most extensive and complex wounds.

In conclusion, the success of any operation for cancer of the head and neck demands an aggressive complete removal of the tumor. It is imperative that the ablative procedure be planned as an integral part of the projected plan for reconstruction.[2] Otherwise, incisions placed without such regard will often eliminate the use of local tissues in the future reconstruction. Too often a patient will survive an extensive operation for cancer to develop fatal complications from the resultant wound. Although local recurrence of cancer is the cause of failure in most head and neck operations, wound problems with resultant hemorrhage, inanition, and pneumonia are certainly the second most frequent cause of failure. The principle of early, complete wound closure still is the *sine qua non* of head and neck surgery.

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A Preliminary Report on "The Pill" and Figure Changes

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Use of "the pill" as a contraceptive has increased until millions of women throughout the world are taking it. The importance of side effects in a drug so widely used is obvious. In a series of 21 women, there was no appreciable change in weight or figure measurements.

TODAY THE "PILL" is probably one of the most widely discussed medications by the laity and the profession. Present day figures show that it is now being taken by millions of women throughout the world.[1] It has proved nearly 100% effective and provides an acceptability of an order higher than that of any previous method of contraception.[2]

The "Pill" has gone beyond being just a prescription filled at the local pharmacy. It has had and probably will continue to have extensive changes in packaging in order to minimize the risk of patients missing a daily dose of the medication and consequently increasing the chances of conception. More and more, we physicians are faced with questions from our patients concerning the possibility of weight gain and/or figure changes, should they embark upon this most effective method of contraception. To the patient with the preconceived idea, usually taken from the lay press or contemporaries, that there definitely will be a change in their body measurements, the answer to this question is very important.

The basis of this evaluation was to determine whether or not there would be any significant changes in either the weight or figure measurements of patients taking a combined progestin-estrogen tablet for contraception.

Method

This study was done on a series of twenty one menstruating patients of varying ages. All patients were selected for this evaluation because of their reliability in following instructions and their willingness in helping solve this problem. Prior to starting on this study, all patients were given a complete gynecological examination and Papanicolau smear. Any patient showing evidence of gynecological disease or positive smear were not accepted into this study. The patients estimate of their two year weight fluctuation, omitting pregnancies, was noted. All patients, prior to this evaluation had not used any systemic form of contraception. Each patient was given a dispensing package of twenty tablets of norethindrone 2 mg. mestranol 0.1 mg., to be taken once daily for twenty days starting on the fifth day of their menstrual period. The first day of their flow was considered to be day one. Each patient reported back to this office monthly to every six weeks while they were on the medication. At each visit they were weighed (in indoor clothing), measure-

ments taken of their chest circumference (undressed), breast cup size (undressed), waist and hip measurements (undressed), and duly noted.

Results

The average weight fluctuation reported for a two year period, omitting pregnancies, was 16.5 pounds. The range was from a four pound fluctuation up to a fifty two pound difference in weight. It was interesting to note that the weight change of all the patients while on the medication fell within the limits of their previous weight histories. Each patent was evaluated for an average of 7.2 months.

Four of twelve patients who gained weight while on the medication started the program at a heavier weight than was reported for their two year previous weight histories. One of the twelve started at a lighter weight than she had been within a two year period and subsequently gained weight on the medication.

Six patients reported a distinct loss in weight while on the medication and three patients had no discernible change in weight.

The menstrual flow in six patients decreased and one had an increase in flow. The remaining fourteen showed no appreciable change in their menstrual flow. Five were heavy bleeders, seven moderate and two had slight flows.

The circumference of the chest increased in fourteen patients, decreased in six and one had no change. The cup measurements increased in five patients, decreased in seven and nine had no change. The waist measurements increased in seven patients, decreased in thirteen and one patient did not change. The hip measurements increased in six patients, decreased in ten patients and five had no change in measurement. See Table I.

Tabulation of Results

Table I

	Number	*Percent*
A. Changes in Weight on Medication		
1. Increased	12*	52%
2. Decreased	6	28.5%
3. No charge	3	19.5%
B. Circumference of Chest		
1. Increased	14	66.5%
2. Decreased	6	28.5%
3. No Change	1	5.%
C. Cup Measurement		
1. Increased	5	24%
2. Decreased	7	33.5%
3. No Change	9	42.5%
D. Waist Measurement		
1. Increased	7	33.%
2. Decreased	13	62%
3. No Change	1	5.0%
E. Hip Measurement		
1. Increased	6	28.5%
2. Decreased	10	47.5%
3. No Change	5	24%
F. Menstrual Flow		
1. Increase	1	5.0%
2. Decreased	6	28.5%
3. No Change	14	66.5%

Discussion and Conclusion

As objective as are measurements in pounds and inches, there are many variables which must be considered in reaching a meaningful conclusion for this preliminary evaluation. The control, the patients two year weight history, was only as accurate as the patient's own recollection of her weight. The indoor clothing with which the patients were weighed in my office varied considerably as to the season, e.g., winter woolens would certainly weigh more than a summer cotton dress. The patients presented themselves to this office at varying times of the day and at varying times in their menstrual cycles. It is a well accepted fact that transient weight changes at any time can be attributable to many extraneous factors, bowel and urinary excretions, psychological stimuli, etc., not actually related to a specific drug therapy. It is hoped that many of these extraneous factors which influence the weights of patients will be averaged out when this study is ultimately completed.

*Includes four patients that started program at heavier weight than normal and one patient at a lighter weight than normal.

Many of the side effects which have been reported with the use of oral contraceptive agents have been most prevalent during the first three months of medication. This preliminary report, along with my personal experience in a large metropolitan area with a busy gynecological practice, have shown thus far that no appreciable changes in weight or figure are discernable in patients taking a regimen of cyclic norethindrone 2 mg. mestranol 0.1 mg. tablets for contraception.

Acknowledgment

Norethindrone 2 mg., mestranol 0.1 mg. was supplied as NORINYL 2 mg. by Mr. Sheppard L. Masarek, Clinical Research Associate, Syntex Laboratories, Inc., Palo Alto, California.

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Monthly Report of Bureau of Communicable Disease Control

	Mar. 1968	Mar. 1967	Jan.-Mar. 1968	Jan.-Mar. 1967
Brucellosis	3	2	3	11
Diphtheria	0	0	0	0
Hepatitis	48	55	126	162
Meningitis (Aseptic)	0	1	0	3
Meningococcal Infections	3	2	15	12
Poliomyelitis	0	0	0	0
Rocky Mt. Spotted Fever	0	0	2	0
Rubella	117	84	222	143
Rubeola	81	413	139	840
Streptococcal Sore Throat (including Scarlet Fever)	2055	1776	5165	4925
Tularemia	1	0	1	0
Typhoid Fever	0	1	3	2
Rabies (in animals)	26	29	61	61
Venereal Diseases				
Syphilis	111	162	380	535
Gonorrhea	788	777	2346	2143
Other	2	0	3	1

The Role of Chemotherapy in the Treatment of Colorectal Cancer

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Chemotherapy with 5-fluorouracil used as an adjuvant to surgery is likely in many cases to control the disease either by preventing further progression or attaining actual regression of metastases. Although cure is not expected, it may be achieved in some cases.

IN OUR PRESENT STATE OF KNOWLEDGE, chemotherapy has no place in the primary treatment of colorectal carcinoma. This is a surgically-approachable disease, and, if possible, should be treated by primary surgical resection. The candidates for chemotherapy are patients who have known or suspected residual neoplastic tissue after surgical therapy, or those who develop metastatic or locally-recurrent disease after what was initially thought to be a curative resection. Included in the group classified as having residual neoplasia are those patients who present with perforative carcinoma of the colon or rectum, regardless of the degree of resectability found possible at the time of the initial surgical attack.

Many different chemotherapeutic agents have been utilized in many different ways in the treatment of colorectal cancer. The alkylating agents such as nitrogen mustard have been administered intravenously[7] with poor results. When used intraluminally at the time of surgery by Rousselot et al.,[14] nitrogen mustard did not improve the results over that of surgery alone. The vinca alkaloids, vinblastine[2] and vincristine,[11] have met with no success, while the antibiotics, such as mitomycin C,[4] streptonigrin[15] and dactinomycin,[17] have had very little better results.

The most effective drugs thus far discovered have been the two pyrimidine analogues, 5-fluorouracil (5-FU) and 2-deoxy-5-fluorouridine (5-FUDR). Response rates from systemic 5-FU therapy have been reported as high as 40%[13] but most studies have yielded about 20% responses.[18,16,1] Studies with 5-FUDR have created some controversy regarding whether it is any more effective than 5-FU,[12] but some reports show response rates to be decidedly better with 5-FUDR.[12,3] The debate is an academic one at the present time since 5-FU is presently being commercially produced and is available for general use, whereas 5-FUDR is still an experimental drug which is in short supply and available only on a restricted basis.

5-FU is potentially quite toxic and especially causes hematological depression and gastrointestinal toxicity. These side effects are particularly profound when the drug is given by intravenous bolus injection, as is recommended in the manufacturers' package insert. A lower dosage schedule, as recommended by Ansfield,[1] has resulted in less toxicity. Others,[5,9,8] have suggested that administration of the drug by an intravenous drip results in even less toxicity while

Presented at the annual meeting of The Medical Society of Virginia, Arlington, October 1967.

achieving results equally as good as the bolus administration method.

Patients who are treated with 5-FU in Adult Cancer Chemotherapy Out-Patient Clinic at the University of Virginia are treated with the lower dosage schedule suggested by Ansfield,[1] which is as follows: 12 mg/kg daily for four consecutive days, then 6.0 mg/kg every other day until the patient develops signs of toxicity or until a *total* of eleven doses have been given, whichever occurs first. Since these are out-patients we have chosen to give them the drug in a 1-2 hour intravenous drip of 5% dextrose water in an attempt to further reduce toxicity. Following the course of eleven treatments, the patient has a rest period of four to six weeks to allow for full recovery from any drug toxicity which might occur, and then the course of treatment is repeated. If, at the end of the second rest period, there is no evidence of progression of disease and no excessive toxicity has occurred, plans are then made for repetitive courses of the drug for an indefinite period, that is, for as long as the response is maintained. This is thought to be necessary because patients have returned after an interval of no drug therapy with recurrent disease which is resistant to 5-FU after having had apparent complete regression of metastases from 5-FU therapy.

The possibility of eradication of disease with drug always exists, although it happens relatively infrequently. However, when a patient shows a good response over a prolonged period on drug therapy, one begins to wonder how much, if any, tumor remains. This quandary ultimately brings one to consider the feasibility of a "second-look" operation. This sort of approach has been followed by Mackman and Curreri[10] in patients given adjuvant 5-FU therapy for one year following resection. Seven of 11 patients who had only palliative resections and 5-FU were found to be free of disease, and three others had only relatively small amounts of residual tumor which were resectable at the second-look procedure. Another 20 patients had "curative" resections but had serosal or mesenteric lymph node involvement at the time of the initial surgery. They were reoperated one year later, after four or more courses of 5-FU therapy, and 17 had no gross tumor detected during re-exploration. Another group of 153 patients who had "curative" resections of the colon or rectum, and were not given adjuvant 5-FU therapy, underwent second look surgery and 70 (45%) had recurrent cancer identified.[6]

On the basis of such findings, we now advocate repetitive courses of 5-FU, as outlined above, for six courses (which should approximate one year) for any patient with residual tumor (known or suspected), as well as those with serosal involvement or tumor-bearing nodes detected in the surgical specimen. At the end of that year, a second look operation should be performed unless the patient has demonstrable disease outside the peritoneal cavity, or unless disease within the peritoneal cavity is obviously beyond resectability.

One other method of utilizing chemotherapy in colorectal cancer should be mentioned and given strong consideration. Rousselot's method of using intraluminal nitrogen mustard (HN_2) with resection was referred to above.[14] This has yielded a four year survival rate of 44% as compared to 48% for the control group which had surgery alone. These were both compared to a group that had intraluminal 5-FU with bowel resection and intravenous 5-FU on post-operative days one and two. The 5-FU-treated group have a very remarkable four year survival rate of 80%!

Considering the data from Rousselot[14] and from Mackman and Curreri[10] perhaps we can project that a combination of these two regimens using 5-FU as an adjuvant to surgery might result in an even greater improvement in the prognosis for the disease.

In closing let me restate what I consider

to be the role of chemotherapy in the treatment of this or any malignant disease. Chemotherapy should not be used with the expectation of curing such patients, although this can occasionally occur. Instead, the chemotherapist hopes to control the disease either by preventing further progression or attaining actual regression of metastases. Even in those patients where control of the disease is not possible, one hopes to at least alleviate the patient's symptoms. The result of the chemotherapist's efforts is intended to be the production of a patient who is at least more comfortable and tolerated by those about him during his remaining life, if not actually to gain a prolonged period of usefulness and productivity for the patient himself.

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Induction of Ovulation

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Before anovulation is considered to be the cause of infertility, several studies including endometrial biopsy, hormone assay, and the effect of hormone administration should be done. Cases of adrenal and thyroid dysfunction should be recognized.

Clomiphene citrate and sequential menopausal chorionic gonadotropin may induce ovulation and result in pregnancy when used in appropriate cases.

THE PRIMARY REASON for attempting to induce ovulation is to treat infertility. Before trying to induce ovulation, therefore, one should be reasonably certain that anovulation is the cause of infertility. Other causes of infertility in the couple should be investigated by determining patency of the fallopian tubes by carbon dioxide or hysterosalpingogram and establishing adequacy of sperm production by a post-coital test (which also provides information concerning the cervical mucus) or with a semen analysis.

The menstrual history may be quite useful in identifying cases of anovulation. Usually, these women will have had amenorrhea or episodes of very irregular bleeding. The history alone cannot be relied upon to exclude anovulation because anovulatory cycles occasionally result in regular monthly bleeding. Basal body temperature recordings over a period of several months are useful when they fail to show a biphasic character. When conducting an infertility investigation, the clinical impression of either ovulation or anovulation should be confirmed by endometrial histology. An endometrial biopsy done on an outpatient basis, or endometrium obtained by uterine curettage will show atrophic, proliferative, or hyperplastic endometrium in instances where the progestational effect after ovulation is not present. Urinary pregnandiol excretion (the excretory product of endogenous progesterone) may also be used. If this value is above 2 mg. per 24 hours, it is assumed that a corpus luteum (and thus ovulation) is present. Under the influence of estrogen without progesterone, the vaginal maturation index will contain many superficial cells while the cervical mucus will become abundant, crystal clear, and will form a microscopic fern pattern when permitted to dry. If ovulation occurs and there is a progestin effect, there will be an increase in intermediate vaginal cells at the expense of superficial cells while the cervical mucus will become scanty and will not form a microscopic fern pattern when permitted to dry.

Good evidence that amenorrhea or oli-

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gomenorrhea is due to anovulation may be obtained by administering an exogenous progestin to a non-pregnant woman, and observing withdrawal uterine bleeding several days after the progestin medication has been stopped. This occurs only if there is endogenous estrogen production. If it is necessary to prime the endometrium with exogenous estrogen before progestin withdrawal bleeding can be obtained, this usually indicates a more severe endocrine disorder than anovulation alone, although anovulation is part of the clinical picture.

To study the cause of anovulation, three endocrine organs should be investigated—the adrenal, the ovary and the pituitary. Adrenal disease should be investigated by determining urinary 17-ketosteroids, and a chemical test of thyroid function such as protein bound iodine should be done. The ovaries may be visualized with culdoscopy or colpotomy and the appearance is often quite helpful in establishing a diagnosis. If endogenous estrogen is low, total urinary pituitary gonadotropins should be determined in an effort to distinguish between a primary ovarian or a primary hypothalamic-pituitary deficit. If the history and general physical examination are suggestive of adrenogenital or Sheehan's syndrome, appropriate diagnostic procedures should be undertaken.

If the above studies indicate that ovulation can likely be induced with thyroid or physiologic doses or glucosteroids (such as prednisone), then such is the most rational therapy. If the diagnosis is polycystic ovary syndrome, one may consider bilateral wedge resection of the ovaries. In most cases, this major surgery is followed for a time with ovulatory cycles, but anovulation may recur. Since the syndrome may be now successfully treated by the medical regimens outlined below, surgery is presently indicated only for patients who understand that it may not cause a permanent resumption of ovulation and do not wish to receive a new drug with potential side effects.

Clomiphene citrate* is an oral synthetic non-steroid compound, related to diethylstilbestrol, but which does not have estrogenic or progestational activity in the human. A rise in FSH and then LH excretion following its administration has been reported and it has been suggested that it is a weak estrogen and competes for binding sites in the hypothalamus and elsewhere, thereby reducing the feed-back inhibition of naturally occurring estrogens.[1] This increase in endogenous pituitary gonadotropins causes the maturation and endocrine activity of the ovarian follicle and subsequent development and function of the corpus luteum. There is also some evidence for direct action of clomiphene on the ovary.[2] In a review of 1888 therapy cycles in 459 women, the ovulation rate was 74% per patient and the pregnancy rate was 21% per patient.[3]

The most common side effect with clomiphene therapy is ovarian enlargement.[4] This is particularly common in patients with polycystic ovary syndrome, and maximal enlargement does not occur until several days after the five-day dose has been given. These cysts spontaneously regress and the ovaries should be permitted to return to normal size before beginning another cycle of treatment. Unless surgical indication for laparotomy exists, this cystic ovarian enlargement should be managed conservatively.

Hot flashes have been reported as other side effects of clomiphene therapy; these are usually not severe and resemble menopausal symptoms. The incidence of multiple pregnancies is increased. Certain patients report abdominal discomfort. This is usually due to mittleschmertz or ovarian enlargement. Ocular symptoms, such as "blurring", were found in a few patients.[4]

The medication should not given to patients with liver disease or a history of liver dysfunction, to patients in which neoplasm

*Clomid, The William S. Merrell Company, Cincinnati, Ohio.

has not been ruled out as a cause of abnormal uterine bleeding, or to women already pregnant.

During the first course of therapy, 50 mg. of clomiphene citrate is taken daily for five days. This can be started at any time in an amenorrheic woman; or if preliminary progestin-induced withdrawal bleeding is desired, the clomiphene should be started on cycle day five. If ovulation does not occur with 50 mg. dosage, 100 mg. daily should be given for five days. If ovulation does not occur after three such treatment courses, it is best to discontinue attempts with this agent. Also, if pregnancy does not occur with three ovulatory cycles, this therapy should be stopped as the safety of long-term treatment has not been proven.

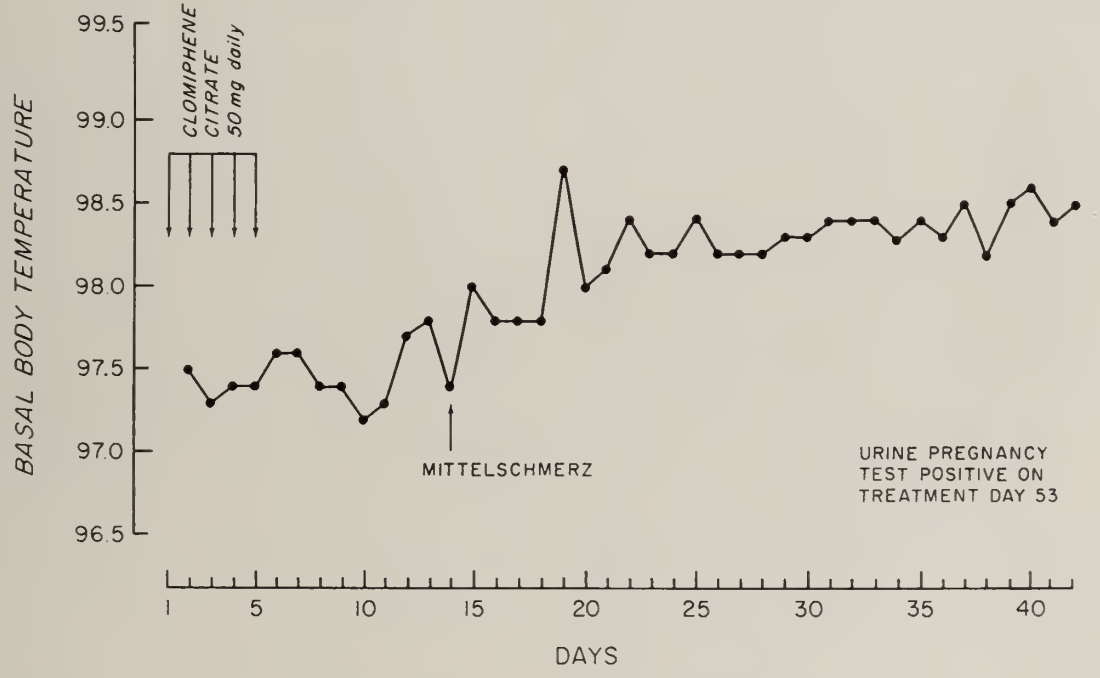


Fig. 1. Basal body temperature chart for Case 1, showing clomiphene therapy followed by ovulation and pregnancy.

Case 1. The patient was a 24 year old woman who had irregular episodes of vaginal bleeding at 18-19 day intervals since menarche at age 11. There was over two years of involuntary infertility. Basal body temperature charts for over one year exhibited no biphasic character.

On physical examination there was moderate facial, breast, and abdominal hirsutism. Breast development was normal and the clitoris was normal. The weight was 116 pounds.

Three endometrial biopsies showed proliferative endometrium. A Rubin's test demonstrated tubal patency and a post-coital test was normal. PBI was normal and urinary 17-ketosteroids were 19 mg./24 hours. Total urinary gonadotropin was between 16 and 50 mouse units/24 hours. The culdoscopic diagnosis was polycystic ovary syndrome.

One attempt at sequential menopausal-chorionic gonadotropin therapy resulted in probable anovulation (urinary pregnanediol was 2.1 mg./24 hours and vaginal bleeding began 12 days after the second HCG injection, but the temperature curve was not biphasic).

Clomiphene citrate then became commercially available; 50 mg. was given daily for five days and a biphasic temperature curve was obtained but pregnancy did not result. This therapy was repeated the next month and during this second cycle of clomiphene therapy a pregnancy occurred (Fig. 1). The

ovaries doubled in size during both cycles of treatment with clomiphene.

Clomiphene achieves its best results in patients with normal levels of gonadotropin and some estrogen production. In other patients and in clomiphene failures, the use of sequential menopausal-chorionic gonadotropin may be considered.[5]

In 1958, Gemzell, et al.,[6] reported that the administration of FSH of human cadaver pituitary origin resulted in ovarian follicular development and estrogen production; when such therapy was followed with injections of human chorionic gonadotropin (HCG) having an LH type of effect, ovulation and sometimes pregnancy resulted. When a proteinaceous extract from the pooled urine of post-menopausal women was found to have high FSH activity, this was substituted for the FSH of cadaver pituitary origin because the raw material was more readily available. The menopausal gonadotropin (HMG) has both FSH and LH activity; most preparations contain primarily FSH.

Twenty women with infertility due to various types of ovulatory failure were treated with sequential menopausal-chorionic gonadotropin by Vande Wiele and Turksoy.[7] Fifteen women conceived one or more times and 12 women were delivered of viable infants. In five of these, the pregnancy was multiple; and in eight of 22 pregnancies an abortion occurred. In a study of side effects associated with such therapy, some degree of ovarian enlargement was noted in 17% of the total series and in 45% of patients who had polycystic ovaries. Marked ovarian enlargement with ascites was seen in 15% of the total series and 6% of the patients with polycystic ovaries.[8]

There are several methods of administering HMG, such as overlapping HMG with HCG therapy, or reducing the dose of HMG for several days before administering HCG.

In the study herein reported, the patients were given daily injections of HMG, beginning at low dosage, and the effect on cervical mucus and vaginal smears noted. The dosage of HMG was increased as necessary to obtain a good estrogenic effect, and when such was obtained, as ascertained by the presence of abundant cervical mucus, injections of HMG were stopped and the patient instructed to have daily coitus. There was one day of no therapy, and then two or four daily injections of 5000 units of HCG were administered. The occurrence of ovulation was documented by basal body temperature charts and by urinary pregnanediol determinations.

Overstimulation was minimized by daily assessment of estrogen activity to detect the optimum time to discontinue HMG and begin HCG administration. Vaginal smears were not helpful in indicating when a good estrogen effect was obtained, but the presence of abundant clear cervical mucus which formed a well developed fern pattern when permitted to dry could be utilized for this. The basal body temperature rise usually occurred one or two days after the second injection of HCG.

In 22 treatment cycles, there was good evidence of ovulation in 18 cycles and four pregnancies resulted. (Table I). There were two instances of probable anovulation in which the pregnanediol was 2.1 or 2.6 mgm. per 24 hours but basal body temperature charts were not biphasic. Some degree of ovarian enlargement was observed in 17 cycles, in two of which the cystic ovary was palpable 19 cm. above the symphysis. There were no cases of ascites.

Case 2. The patient was a 26 year old gravida i whose only previous pregnancy had been an ectopic tubal pregnancy, and who had two years of involuntary infertility. Menses began at age 12, and occurred irregularly every 2-3 months. Physical examination disclosed normal findings.

A Rubin's test indicated tubal patency and a post-coital test showed adequate sperm. A biopsy showed proliferative endometrium and basal body temperature

TABLE I

SEQUENTIAL MENOPAUSAL—CHORIONIC GONADOTROPIN FOR OVULATION INDUCTION

Case Number	Patient's Age	Recent Menstrual History	Ovaries	Maturation Index (Parabasal-Intermediate-Superficial)	Endometrium	Total Urinary Gonadotropin (mouse u/24 hrs.)	Therapy FSH (units)	Therapy LH (units)	Therapy HCG (units)	Results	Ovarian Enlargement
1	24	oligomenorrhea	culdoscopy: polycystic ovaries		proliferative	16–50	880	248	10000	probable anovulation (pregnanediol 2.1)	x 2
2	26	oligomenorrhea	culdoscopy: polycystic ovaries	0–88–12	proliferative	6–16	600	12	10000	ovulation	6 cm
							600	12	10000	ovulation	x 3
							1190	85	10000	ovulation	x 3
							1120	316	10000	ovulation	8 cm
3	21	oligomenorrhea	colpotomy: polycystic ovaries	0–91–9	proliferative	6–16	975	325	20000	ovulation	0
							900	300	20000	ovulation	x 2
							1200	23	10000	ovulation	x 2
							1125	21	10000	ovulation	x 2
							1400	100	10000	pregnancy	0
4	23	oligomenorrhea	culdoscopy: polycystic ovaries	0–52–48	proliferative	<6	375	125	20000	anovulatory cycle	0
							1050	350	20000	pregnancy with spontaneous abortion at 4 months	19 cm above symphysis
							2240	160	10000	pregnancy	10 cm.
5	21	oligomenorrhea	culdoscopy: polycystic ovaries	0–94–6	proliferative	6–16	975	325	20000	ovulation	x 3
							900	300	10000	probable anovulation (pregnanediol 2.6 mg. /24 hours)	0
6	30	amenorrhea	culdoscopy: polycystic ovaries	0–40–60	hyperplasia	6–16	1950	38	10000	ovulation	0
7	27	oligomenorrhea	previous wedge resection: polycystic ovaries	0–90–10	proliferative	<6	1050	350	10000	ovulation	8 cm
							560	12	10000	ovulation	x 2
							770	55	10000	anovulatory cycle	x 2
							880	248	10000	ovulation	5 cm
8	23	amenorrhea	culdoscopy: polycystic ovaries	0–85–15	proliferative	<6	1400	100	10000	pregnancy	8 cm
9	30	oligomenorrhea	culdoscopy: polycystic ovaries	0–100–0	proliferative	<6	450	150	20000	ovulation	19 cm above symphysis

charts were not biphasic except for one apparently isolated spontaneous ovulation on the day of culdoscopy. The culdoscopy demonstrated polycystic ovaries. PBI was normal and urinary 17-ketosteroids were 2.1 mg./24 hours. Total urinary gonadotropins were between six and 16 mouse uterine units/24 hours.

The patient received sequential menopausal-chorionic gonadotropin for four cycles and apparently ovulated each time, but did not become pregnant. The fourth cycle is shown in Figure 2. Since this therapy, she has received three cycles of clomiphene therapy and has apparently ovulated two times without becoming pregnant.

A Rubin's test showed tubal patency and a post-coital test showed adequate sperm. Basal body temperature curves were not biphasic and endometrial biopsy contained proliferative endometrium. Thyroid function studies were normal; total urinary gonadotropins were between six and 16 mouse uterine units/24 hours. At exploratory colpotomy, ovaries were twice normal size with smooth white capsules, and the diagnosis of polycystic ovary syndrome was made.

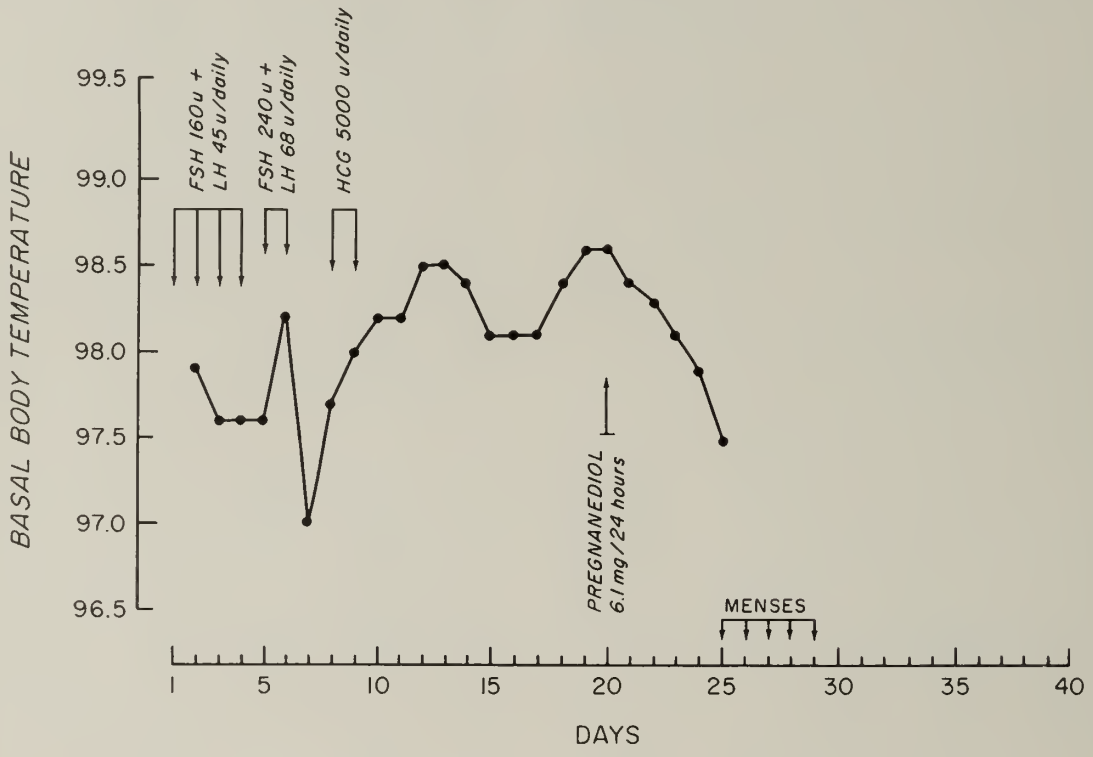


Fig. 2. Basal body temperature chart for Case 2, showing sequential menopausal-chorionic gonadotropin therapy followed by ovulation but not conception.

Case 3. The patient was a 21 year old gravida o who underwent menarche at age 11 followed by irregular menses since age 15. When first seen she had had four years of involuntary infertility. Physical examination was not remarkable except for a small amount of hair above the upper lip.

The patient received sequential menopausal-chorionic gonadotropin for four cycles with apparent ovulation (as determined by basal body temperature and urinary pregnanediol excretion). Following the fifth cycle of therapy she became pregnant (Fig. 3).

Summary

One cause of involuntary infertility is anovulation. Instances of thyroid and adrenal dysfunction should be recognized. Anovulation associated with normal endogenous

estrogen levels (such as is often the case in polycystic ovary syndrome) may be treated with clomiphene citrate with a 74% incidence of ovulation and a 21% incidence of pregnancy. In cases where the estrogen levels are low or in clomiphene failures, injections of sequential menopausal-chorionic gonadotropin may induce ovulation and result in pregnancy. The major side effects of both types of therapy have been ovarian cysts, multiple pregnancy and abortion.

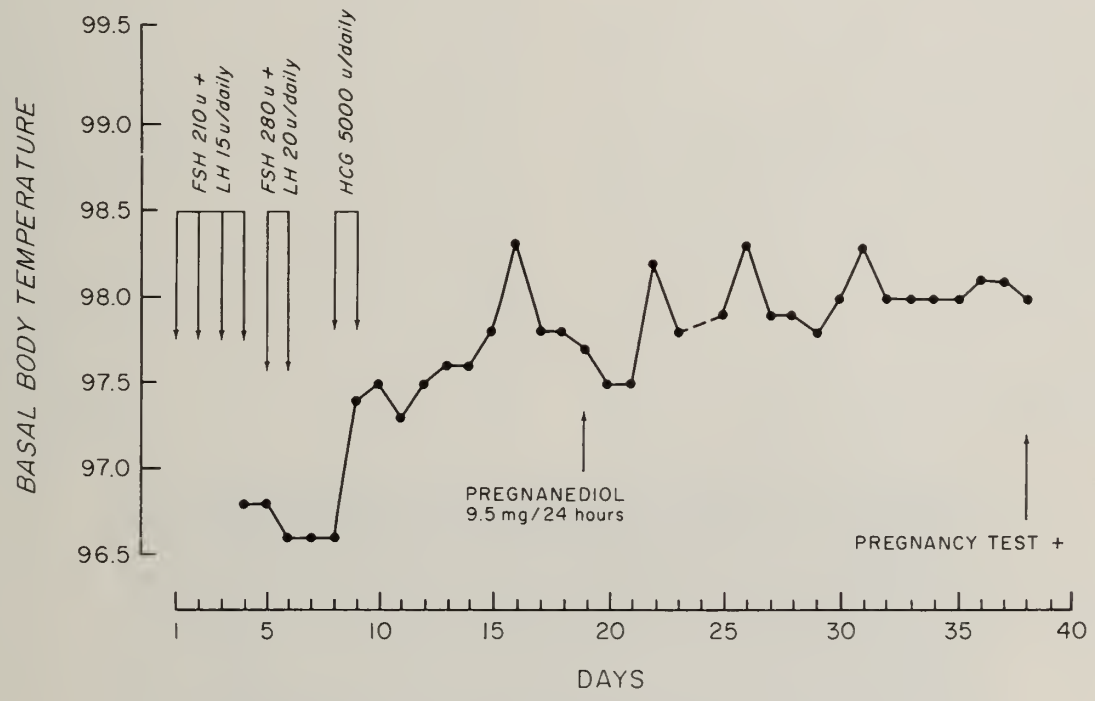


Fig. 3. Basal body temperature chart for Case 3, showing sequential menopausal-chorionic gonadotropin therapy followed by ovulation and pregnancy.

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Clinicopathological Conference . . . .

Abdominal Catastrophe Complicating Plasma Cell Leukemia

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Clinical History

A 54 year old female was brought to the emergency room of the Medical College of Virginia Hospital at 4:00 A.M. on 3-29-67 because she felt "deathly sick".

A diagnosis of multiple myeloma with plasma cell leukemia had been made at another hospital in September, 1963, on the basis of multiple osteolytic bone lesions, anemia, thrombopenia, and peripheral white count of 55,400/mm^3 with 91 percent plasma cells, Bence-Jones proteinuria and a serum protein of 11 gm. percent with 6.6 gm. globulin. There was a marked increase of gamma globulin on the electrophoretic pattern (fig. 1) indicative of multiple myeloma. A marrow aspirate consisted chiefly of abnormal plasma cells. With a BUN of 48 mg. percent and severe respiratory dysfunction secondary to multiple rib fractures, she was begun on prednisone and melphalan ("Alkaran"). She was followed in the hematology clinic at the Medical College of Virginia where this regimen was continued. Her hemoglobin rose from 7.6 gm. percent to 12.1 gm. percent over a period of four months with a decrease in peripheral plasma cell count, a rise in platelet count and a decrease in BUN. Her respiratory difficulty improved markedly and she was able to do light housework. She required hospitalization in October, 1966, because of a drop in her hemoglobin, and a rise in BUN (112 mg. percent) and uric acid (15.9 mg.). Her chemotherapy was interrupted



Fig. 1. The patient's serum contains a large quantity of abnormal globulin (right, upper). The lower strip is from a normal control.

for several weeks and she was treated vigorously with fluids, alkalinization of her urine, and allopurinol. She required five transfusions during this time but her hemoglobin appeared to stabilize, the white count and platelet count rose to normal levels and her BUN fell to 25 mg., and the serum uric acid to 2.3 mg. percent. The prednisone and melphalan were reinstituted and she was discharged. She continued to feel well but her hemoglobin concentration remained between 7-8 gm. and her platelet count hovered between 50-100,000/mm^3. She had no abnormal bleeding except at the site of venipuncture but it was felt that the melphalan mustard might be playing a role in her platelet depression. Because of this she was changed over to cyclophosphamide in February, 1967. She was seen in the clinic on 3-28-67 stating that she was much weaker and quite nauseated but with no new physical findings. Hemoglobin was 6.1 gm., WBC 11,000/mm^3 with 76 percent plasma cells, 8 percent neutrophils, and 16 percent lymphocytes. Platelets 18,000/mm^3.

In the emergency room she was acutely dyspneic but conscious and oriented. Vital signs included BP 0/0, P. 100, T. 97°, R. 30. She was given Ringer's lactate intravenously with a slight rise in blood pressure and a further improvement after transfusion of whole blood. She was quite pale. There were numerous large hemorrhages in the left fundus. Lungs were clear. The heart was of normal size. There was a tachycardia with a regular rhythm. A grade III systolic murmur was heard over the entire precordium. The abdomen was slightly distended, but no organs or masses were detected. Bowel sounds were present and normal. Shifting dullness was present. Peripheral pulses were normal. Pelvic examination was negative.

Laboratory data: Hemoglobin 3.9 gm., WBC 22,000/mm^3 with 62 percent plasma cells, 24 percent neutrophils and 19 percent lymphocytes. Urine: specific gravity 1.017, 3+ glycosuria, positive for protein and blood, negative for acetone. Microscopic examination revealed 50 RBC/hpf, 12-20 WBC/hpf and occasional coarsely and finely granular casts. BUN 30 mg. percent, blood sugar 550 mg. percent, serum sodium 125 mEq/1, serum potassium 6.1 mEq/1, chlorides 101 mEq/1, and CO_2 less than 5 mEq/1.

A chest x-ray revealed slight cardiomegaly but was otherwise negative. Flat plate of the abdomen was reported as negative.

An ECG revealed a sinus tachycardia with a shift of the frontal plane axis to the right compared to a previous tracing on 10-21-66. There were Q waves of questionable significance in leads II, III, and AVF. Subsequent tracings failed to show progression of these findings.

Gastric aspirate was faintly positive for blood. The admission stool specimens were negative for blood but became quaiac positive by 10:00 A.M., 3-29-67. Because of abdominal distention and shifting dullness peritoneal aspiration was done in left lower quadrant. This produced dark blood which failed to clot.

Patient was treated with fresh whole blood, platelet rich plasma and intravenous fluids. Her condition remained critical with fluctuating blood pressure. On 3-31-67 an exploratory laparotomy was performed.

Clinical Discussion

Dr. Robert B. Scott (Assistant Professor, Department of Medicine):

Our patient had an approximately one year and a half course of plasma cell leukemia and multiple myeloma. These diagnoses were based upon leukocytosis, many abnormal plasma cells in the peripheral blood, anemia, osteolytic bone lesions, urinary Bence-Jones protein, a bone marrow containing largely abnormal plasma cells, and a high serum globulin with the typical spike on the electrophoretic examination. Let me show you from the peripheral blood smears of this patient the unusual finding of many plasma cells. Many are of typical plasma cell appearance; others simulate lymphocytes or

the so-called "plasmacytoid lymphocytes". As we look at the bone marrow we note that the great preponderance of the white cells is abnormal plasma cells (fig. 2). It is noteworthy that platelets were only rarely seen in these smears. It is not surprising from the morphology of this marrow that the patient had anemia and thrombopenia. We see on the electrophoretic pattern determined from the serum a striking myeloma spot. Another important feature compatible with myeloma is that the rest of the gamma globulins are decreased.

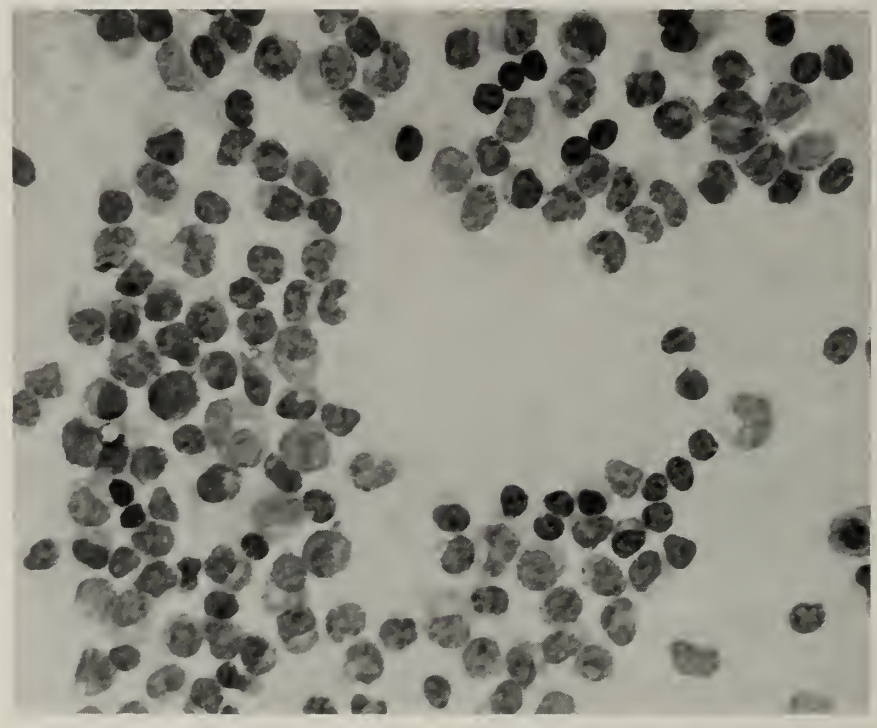


Fig. 2. The bone marrow aspirate consisted largely of abnormal plasma cells and "plasmacytoid lymphocytes".

As is so common with myeloma the patient was severely ill when she presented and when the diagnosis was made. The prognosis is especially bad when there is renal disease apparent at the time of diagnosis. This lady's original blood-urea-nitrogen was 48 mg. percent. She was begun on treatment with our current standard therapy of corticosteroids and L-phenylanine mustard. The generic name of the latter drug is melphalan, the trade name is Alkaran. She showed a good initial response and I had the opportunity of seeing her in our Blood and Spleen Clinic. She got what we thought was a good response, tending to refute the grim attitude about myeloma that most of us have. Perhaps 20 to 25 percent do get a good response to treatment. Her response was noticable in a period of a few weeks. She used to come by ambulance to the clinic where she was brought in on a stretcher complaining of her bone pain. She was very anemic and thrombopenic. Over a period of weeks she got better and better and was able to come to the clinic in an automobile and to walk into the building. Her pain disappeared; she was able to do her housework. Her good response was maintained for many months by the drug until she was hospitalized later because of a rise in her BUN indicating impending renal failure. With increased dosages of the melphalan, she got much better. She had no abnormal bleeding during this time except at the site of venipuncture. This was despite a platelet count of only 50,000. Patients do not readily bleed unless their platelets are below 30,000 unless there are other factors.

On the evening before her final admission

she became very ill and was brought to the emergency room dyspneic and with an unobtainable blood pressure. Despite some apparent dehydration her hemoglobin was 3 gm. lower than that of the day before when checked in the clinic. We began our search for the assumed bleeding. A small hemorrhage was noted in the left fundus. This was of no particular significance at this point because many patients with severe anemia have hemorrhages in the eye grounds without a bleeding diathesis. In this patient, however, it may have been related to a low platelet count. The heart size was normal in spite of a chronic anemia. The murmur, I think, we can relate to her hemoglobin which was now down to 3 gm. Pelvic examination was unremarkable. Her peripheral pulses were good following transfusions and fluid infusion. No petechiae or other bleeding manifestations were noted until examination of the abdomen showed the presence of shifting dullness and that the abdomen was slightly distended. She had mild-to-moderate generalized pain which did not seem to localize in the abdomen. Could we see the x-ray at this point, Dr. Neal?

Dr. M. Pinson Neal, Jr. (Chairman, Division of Diagnostic Radiology):

The first films I have available date to October, 1966, approximately one year following her original diagnosis. The typical multiple punched out lesions seen in multiple myeloma are noted in the ribs and in the proximal end of the humerus. Some are expansile. The more typical are the punched out lesions without reaction or sclerosis about them.

The next films were taken during her final admission late in March, 1967. The pelvis shows the punched out lesions of myeloma as well as demineralization and osteoporosis. This film shows some gas distended bowel in the central abdomen. This was taken at the time when the shifting dullness in the abdomen was noted clinically. The abdominal density is homogeneous. We see no psoas shadows and so suspect intra-abdominal fluid. According to the history, blood was obtained from the abdomen by paracentesis, yet we see no reaction in the form of the ileus we commonly expect with intra-abdominal hemorrhage. The upper gastrointestinal series of films was not remarkable. The chest film shows that the punched out lesions have increased only a little bit in size in the previous eight months. The heart is also slightly larger.

The last group of films was taken shortly before death and show a tracheostomy tube in position but no dramatic changes in the chest. The bony lesions are still consistent with myeloma though metastatic carcinoma cannot be completely ruled out. Changes indicative of ascites are noted in the pelvis. No free air is found under the diaphragm.

Dr. Scott: The gastric aspirate was said to be faintly positive for blood. This, to me, has little significance in view of the effect of even a mild trauma to a patient with a low platelet count. Passing a tube may produce a faintly positive gastric aspirate in many normal people. The initial stool specimen was negative for blood but the stool became positive later. Perhaps this lady was having an acute intestinal bleed. More suggestive findings, however, are those of the shifting dullness, the abdominal distention, and the aspiration of dark blood which failed to clot. Blood in the peritoneal or pleural cavities often does not clot when taken out because it has been defibrinated. The fibrin becomes layered out on the surfaces of the cavity aided by body movement and leaves the red blood cells in a solution of serum. So our little story ends with the patient receiving transfusions of fresh blood and being taken to surgery presumably to explore what appeared to be an intra-abdominal hemorrhage.

Let me make some general remarks about myeloma and related diseases. Myeloma is considered one of the paraproteinemias, that is, one of the diseases associated with the presence of an abnormal protein in the

blood. Franklin[1] reported approximately 400 patients who were found to have abnormal protein in their serum. Sixty-five percent of these patients had myeloma. Macroglobulinemia, presumably Waldenstrom's disease, accounted for ten percent. Malignant lymphoma with a monoclonal gammopathy accounts for six percent. The latter are lymphosarcomas in which the abnormal lymphoid cells do not look like plasma cells but nevertheless produce a serum protein closely resembling that seen in myeloma. Some of these may represent variants of yet to be described disorders. Heavy chain disease or Franklin's disease is the association of lymphoma with the production of the heavy chains of the gamma globulin molecules and with diminished or absent light chain gamma globulin production. The heavy chains appear in the urine in the same way the light chains such as Bence-Jones proteins appear in the urine in myeloma. Some patients with carcinoma or other non-lymphomatous neoplasms may develop a relatively homogeneous spike in their gamma globulin region. Some people have the same spike on their electrophoretic pattern without apparent disease. This is sometimes termed benign monoclonal gammopathy. It remains to be seen whether these patients are going to have myeloma or malignant lymphoma at some later stage or not.

I have variously referred to the patient's condition today as plasma cell leukemia and as multiple myeloma. The two are part of a spectrum. At one end is the solitary osseous or solitary extramedullary plasmacytoma and at the other end a frank plasma cell leukemia. Most patients, perhaps two-thirds of them, fit somewhere in the middle with what we know as multiple myeloma. A diligent search in the blood of patients with multiple myeloma will show a few abnormal plasma cells in perhaps thirty percent. Many more have abnormal plasma cells in their peripheral blood terminally. Perhaps in this patient we can explain the leukemic picture by saying that her malignant cells possessed abnormal membranes which had characteristics permitting the cells to be released from the marrow and to float free in the peripheral stream as opposed to being struck together as a plasma cell tumor or held in the marrow by other membrane phenomena.

One of our patient's major problems was renal disease. Myeloma of the kidneys is a disastrous complication for many patients. It is related, if not due, to depositions of the abnormal or Bence-Jones protein in the kidney. Hypercalcemia may occur and produce nephrocalcinosis. Myelomatous infiltration may infiltrate the kidney physically but it is uncertain whether or not this can produce frank clinical renal disease. Patients with myeloma have been reported to have hypophosphotemia, uric acid in excess in the urine, phosphate excess in the urine, glycosuria, and renal tubular acidosis. We cannot be sure that our patient had any of these. There is some suggestion that she might have from the fact that her chloride was 101 at a time when her sodium was 125. We don't know what part azotemia and hyperventilation played in the development of a low CO_2. It is possible that she had a renal tubular acidosis. Amyloid in the kidney may have contributed to her renal impairment. Amyloidosis occurs in about 10 percent of patients with multiple myeloma. Its distribution is rather like that termed primary amyloidosis with macroglossia, cardiomegaly, and infiltration of other organs.

The patient's final episode appears to be associated with bleeding. Why do patients with myeloma bleed? Significant thrombocytopenia is almost inevitable in this disease. It may become impossible to find a megakarocyte in the marrow or a platelet in the peripheral blood. There are, however, other reasons why people with myeloma may bleed. In some of these patients and in some others with abnormal globulin the abnormal protein interferes physically with the polymerization of fibrin and with some of the

other clotting factors as well. Some patients have been shown to have an increased fibrinolysis asociated with their myeloma proteins. A heparin-like anticoagulant in the circulating blood has been described.[2] These disorders, however, are usually associated with a generalized bleeding tendency and not as in our case with specific sites of blood loss.

We want to know specifically what was the cause of her intra-abdominal hemorrhage. We have no history of trauma. The only one I can relate to her disease might be possible rib fractures lacerating an organ such as the liver or spleen. She could have had such trauma from someone lifting her out of bed or otherwise moving her about. We have no evidence of unassociated causes of bleeding. So-called "abdominal apoplexy" can produce intra-abdominal hemorrhage and is believed to be due to arteriosclerotic vessels bursting in the abdomen. Hemangioma of the liver, spleen, or intestine may bleed producing spontaneous intraperitoneal hemorrhage but we have nothing on which to make that diagnosis. In younger women we might wonder about a rupture of a Graafian follicle or about a tubal pregnancy. One factor to be considered seriously in obscure bleeding in a myeloma patient is that, at least in autopsy material, about twenty percent of these patients have an additional neoplasm. There are usually intestinal neoplasms such as carcinoma of the colon. I shall worry a little bit about this lady having a second tumor such as the colon cancer which might be bleeding into the abdominal cavity. There is nothing else, however, to suggest this. Spontaneous rupture of the liver or spleen is known to occur when there is lymphoma infiltrating these organs.[3] The process and infiltrate of infectious mononucleosis can also produce rupture of these organs.[4] The amyloid-involved spleen or liver can rupture. As our patient is somewhat predisposed to amyloid, this is a rare possibility to be considered.

I do believe the patient suffered a spontaneous rupture of an abdominal organ. The liver and the spleen are those most likely. Perhaps one of these was altered by amyloid or leukemic infiltration.

Ward Diagnosis:

1. *Plasma cell leukemia.*
2. *Abdominal hemorrhage, ? spleen.*
3. *Gastrointestinal bleeding.*

Dr. Scott's Diagnosis:

1. *Plasma cell leukemia.*
2. *Amyloidosis, secondary to (1).*
3. *Rupture of abdominal organ, liver or spleen.*

Pathological Discussion

Dr. Charles L. Johnston (Associate Professor in Clinical Pathology):

The surgeons found an estimated 2,000 ml. of blood in the peritoneal cavity. Splenic tears were noted with oozing from the surface of the spleen. The organ was removed. The total blood loss at operation was estimated at 4,200 ml. Examination of the spleen in the Surgical Pathology laboratory revealed that the spleen was only slightly enlarged at 380 grams. Three or four capsular tears, ranging to 1 cm. marred the surface. The pulp grossly was dark red and of firm consistency. The architecture was obscured. Through the microscope we saw that the splenic architecture was replaced by atypical plasma cells. They infiltrated the vessels by subendothelial dissection, replaced the follicles and pulp, and obliterated the trabeculae. The capsule was densely infiltrated in many areas and plasma cells and blood could be seen extending out into the adjacent fat in the areas of the hemorrhage (fig. 3).

On the first day after surgery the patient's hemoglobin and hematocrit were stable; however, she did poorly because of congestive heart failure and poor ventilation. In spite of the tracheostomy, she developed, on the second day, a Cheyne-Stokes

respiration. Her blood pressure became unobtainable. The platelet count which had been about 50,000 prior to surgery fell to levels of 10,000 to 20,000. On the third day following surgery the patient developed generalized seizures and died.

At autopsy there was a massive subarachnoid hemorrhage which was probably the immediate cause of death and which probably explained the seizures. This is not an uncommon complication of various bleeding diathesis including thrombocytopenia. The heart was mildly enlarged as Dr. Neal suggested but showed only superficial epicardial ecchymoses. Numerous small ulcerative areas were present in the stomach. The liver was somewhat enlarged and had a mottled appearance as is seen so often in leukemias. The small amount of free blood in the peritoneal cavity did not suggest a re-bleed following splenectomy. The bone marrow, particularly the vertebrae, showed numerous destructive lesions with mucoid gelatinous material replacing the usual trabecular appearance. Microscopically the marrow was almost totally replaced by the abnormal plasma cells. These were seen as light-to-moderate infiltrates in many tissues. The infiltration was particularly dense and diffuse in the liver. Relative to the renal problems, a moderate infiltrate of plasma cells was seen in the kidney. The tubules were vacuolated and contained some dense proteinacious material. No evidence, however, of amyloid was found in the kidneys or elsewhere.

I prefer the term plasma cell leukemia for this patient's course rather than multiple myeloma. This is based on the classic criteria for plasma cell leukemia noted by Askanazy and Dubois-Ferriere.[5] These include leukocytosis, diffuse bone marrow involvement as opposed to isolated osseous myeloma tumors, and presence of plasma cells in the peripheral blood (more than just terminally). To these are usually added the feature of signs and symptoms of acute, subacute, or chronic leukemia. There are several differences between patients with plasma cell leukemia by these standards and multiple myeloma. There is no sex predilection among the leukemic patients whereas multiple myeloma



Fig. 3. The leukemic infiltrate extends from the splenic pulp into vessel walls, splenic capsule and adjacent adipose, presumably weakening the splenic capsule.

is more common in males. The age range is about the same, upwards of 50 years. The average duration of the disease has been six to eight months for the leukemia and about two years for the multiple myeloma patients.[2] Both figures are being altered by our current methods of treatment.

You might wonder about the incidence of plasma cell leukemia. In humans it is the rarest form of leukemia. The ratio of myeloma to plasma cell leukemia is about 50 of the former to one of the latter. After reviewing material from this institution it appears that we see at least 250 cases of acute leukemia for every one of plasma cell leukemia. Spontaneously occurring plasma cell leukemia is somewhat more common in other species of animals.

Splenic rupture associated with leukemia was reviewed in 1966 by Stites and Ultman.[6] They reported 32 cases and added one of their own. Only three of these cases were in females. Twenty-three had moderately adequate descriptive detail and of these, ten met the criteria of acute leukemia. Five of these were myeloid, four lymphatic and one undifferentiated. There were nine chronic myeloid leukemias, three chronic lymphatic leukemias and one lymphatic leukemia not otherwise classified. I have been unable to find, in a rather thorough search, a previously reported case of plasma cell leukemia and splenic rupture. For that matter, splenic rupture with multiple myeloma has not apparently been reported. It is fair to say then that we are dealing with an extremely rare complication of a very rare disorder.

Dr. John H. Moon (Associate Professor in Medicine):

Plasma cell leukemia has not been quite so rare in our experience as Dr. Johnston's figures would indicate if one defines the disease as an elevated total white count with a preponderance of plasma cells in the peripheral blood. We have had five cases in the past half dozen years, an incidence of about one in forty cases of myeloma that we see. Patients referred to medical centers tend to be those who are more complicated and otherwise unusual. I suspect that is why our incidence is higher than the average.

Dr. Scott: Though splenic rupture with plasma cell leukemia may be unique, this complication of other leukemias and lymphomas is not so unusual. The incidence will increase as we prolong the course of these diseases with more effective therapy. We must diagnose or anticipate the rupture. Splenectomy can be, and has been, performed in these patients with relatively low morbidity and mortality.

Final Anatomical Diagnoses:

1. *Plasma cell leukemia, involving spleen, liver, bone marrow and other tissues.*
2. *Rupture of spleen.*
3. *Thrombocytopenia, secondary to (1).*
4. *Subarachnoid hemorrhage, secondary to (3).*

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Glue Sniffing: A New Symptom of an Old Disease

"Glue sniffing" continues to be a frequently encountered form of aberrant behavior among modern adolescents. The first recognition of the problem appeared in the literature in 1959, but undoubtedly the practice had been going on for some time prior to its recognition by medical authorities. There have been at least nine deaths in the United States attributed to this practice, one of which occurred in Richmond, Virginia, in February, 1963.

Method

The glue sniffer aspires to achieve an euphoric escape from reality by inhalation of organic solvents contained within plastic cements. He may squeeze the contents of a tube of plastic cement onto a handkerchief or into the bottom of a paper or plastic bag. The cloth or bag is then held over the nose and the vapors inhaled until the desired effects are obtained. The glue may be placed on a heated pan for more rapid effects.

Effects

The desired effects are those of acute alcoholic intoxication with feelings of exhilaration, being "drunk", "dizzy". Many have described vivid dreams, often in color, or hallucinations. The sniffer's speech is slurred. He becomes depressed, mentally confused and may display bizarre behavior, or may black out.

The glue sniffer's "euphoria" with feelings of reckless abandon often leads to dangerous impulsive acts. One boy in his early teens is reported to have assumed a fighting stance before an oncoming train and narrowly escaped death. Another young glue sniffer, convinced he could fly, leapt to his death from the top of a building.

Sniffing is usually followed by group horseplay, fighting and in some instances homosexual activity. They often commit other misdemeanors while under the influence of glue vapors, i.e., breaking and entering, petty thefts.

Prevalence

A statistical description of the problem is not necessary to appreciate its existence. None of the studies conducted thus far are conclusive; however, glue sniffing appears to be more prevalent in the nine to 16 year-old-age group with a greater incidence among boys than girls. It is usually practiced in gangs with group meetings in secluded places away from parental control.

Ethnic Backgrounds and Personality Patterns

The earlier reports on glue sniffing indicated that this was a practice primarily of racial minorities and children from low socio-economic backgrounds. The more recent articles indicate that the fad is spreading to include children from all sectors of society.

A substantial number of children studied have other habits, such as smoking or imbibing alcohol and in one study, alcoholism was a trait of at least one parent in 37% of the families studied. Family backgrounds studied have shown general instability characterized by the absence of a parent from the home for a number of years during the child's lifetime. As a result, the child suffers from severe emotional deprivation and

insecurity with both his family and his peer group.

A study of 19 adolescent glue sniffers by Brozovsky in 1965 revealed this habit was practiced in an attempt to replace feelings of helplessness and depression. The imbibing of alcohol by one-half of these children and the presence of chronic alcoholism in their immediate families point to a conducive environment for later habituation to alcohol and possibly to drugs.

Tolerance

Although an increasing tolerance to the amount of vapor inhaled occurs with repeated bouts, true addiction has not been found. The emotionally well-balanced and well adjusted child might experiment once or twice with glue sniffing as a result of a combination of curiosity, boredom and availability, but he would not be likely to indulge in repeated bouts.

Toxicity

The problem of chronic toxicity resulting from repeated inhalations of the fumes of plastic cements is difficult to assess. There is a discrepancy in reported toxic manifestations with scant scientific documentation of pathologic effects. There is no proof of irreversible damage to the brain, liver, kidneys, or bone marrow. Glue sniffing is physically harmless to most children. Potentially serious toxic effects can occur, however, as exemplified by the reported nine deaths.

Treatment

The treatment of acute poisoning resulting from glue sniffing is not specific but only supportive. Probably it should be handled in a similar fashion as the acute intoxication resulting from alcohol intake.

Solutions

Several cities and states (including Virginia) have passed ordinances restricting the sale of glue or making glue sniffing illegal. However, legislation may fail in preventing promiscuous consumption or fail in stopping the wanton sales of dangerous products. Abolition of this very useful product will not help because there are many other products such as gasoline and cleaning fluids that could soon become a substitute. However, the Virginia law is specific in banning the inhalation of other vaporous substances in the place of glue.

Virginia legislation prohibits the deliberate inhalation of noxious susbtances for the purpose of producing ". . . conditions of intoxication, inebriation, excitement, stupefication or dulling of the brain or nervous system."

Persons violating this law are subject to fines up to two-hundred dollars and/or jail sentences not exceeding six months.

The City of New York has enlisted the aid of industry in using safer or alternative solvents in the manufacture of glue. They have also introduced factual material about glue sniffing in the curriculum content at the elementary and high school level.

Dr. Edward Press, past editor of the Journal of Pediatrics, recommends a "coordinated approach" as follows:

(1) Correct the underlying emotional disorder when this exists.

(2) Educate parents and children to the potential risk of glue sniffing.

(3) Make glues and related substances less available on the open "serve yourself" shelves of stores.

(4) Substitute less toxic organic solvents wherever possible.

(5) Add special "odor retardants" to these substances.

The problem of glue sniffing among adolescents is prevalent enough to warrant consideration by the physician of the effects of its unrestricted use on the individual and the community. In cooperation with other community and social groups, the physician should be prepared to contribute to the control of this practice. Glue sniffing is indeed a new symptom of an old disease—the social and emotional deprivation of children.

Mental Health....

JOHN B. DYNES, M.D.

Lobotomy—Twenty Years Later

Frontal lobotomy was employed as a form of treatment for a variety of mental disorders, as well as a surgical procedure for the relief of intractable pain, and was widely used in the post World War II period, but has been employed only rarely in the past ten years. Hope held high at the time by both research workers and clinicians that an effective treatment for mental disorder had been found. Frontal lobotomies were done by the thousands around the world. A spate of papers were published, to which I plead guilty of contributing my small bit.[1,6] Now, some twenty years later the "tumult and the shouting" has subsided but "lest we forget" the lessons learned, I have undertaken this clinical study.

John Fulton[2], in his Salmon Lectures in 1950 gives an interesting and informative account of the research that led up to and followed the first use of lobotomy by Moniz in 1935, in the treatment of mental patients. A number of books and extensive research projects were published[3,4,5,6,7] and may be referred to by those interested. Today every major mental institution is probably confronted by a group of lobotomized patients. I have had the opportunity of assessing such a group of 83 patients at present confined to the VA Hospital in Salem, Virginia. This group represents the poor end results of this form of treatment. This report is in no way intended to be a criticism of those who recommended the operation, nor of the surgical method employed.

The pre-operative diagnosis in 82 of the 83 patients was schizophrenia. The 83rd patient was diagnosed as a chronic brain syndrome. Great care had been taken in selection of these patients. The mental disorder had been present in all patients for at least two years and in the majority for more than five years. All patients had either been episodically violent and difficult to control, or had shown marked agitation or assaultive behavior, along with delusions, hallucinations and inappropriate affect, the hallmark of the schizophrenic disorders. Seventy-nine or 95% of the group had previously had either electroshock therapy or insulin shock therapy, as well as a variety of sedative drugs, hydrotherapy, psychotherapy, occupational therapy, total push, all without improvement.

Without exception all patients were temporarily improved following lobotomy, making nursing care much easier. This improvement with lessening of violence and restlessness lasted from a few days to a few months and in most patients there was a persistent lessened tendency to violent behavior. In none of these patients was there any appreciable change in their mental disorder. Delusions and hallucinations and unpredictable episodes of restlessness and at times violence continued to occur. If anything, there seemed to be a hastening of the process of deterioration seen in most chronic schizophrenics. The inappropriate emotional response with withdrawal, lack of insight and flattening of affect with irresponsibility and failure of social adaptability, as well as intellectual deterioration were the chief features that kept these patients in the hospital.

From the clinical standpoint it may be difficult to separate the "burned out" or deteriorated schizophrenic from the chronic brain syndrome group, but after my examination of each patient, I would conclude

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Approved for publication by the Commission, Department of Mental Hygiene and Hospitals.

that we must now add the diagnosis of chronic brain syndrome to the diagnosis of schizophrenia in most of these lobotomized patients. This was anticipated in reports of the Third Research Conference on psychosurgery[7] in 1951, at which it was reported that 25% of patients following lobotomy had evidence of intellectual impairment. The degree of intellectual impairment seemed to be correlated, in part at least, to the type of operative procedure employed. In this series of 83 patients, 80 had the standard or total frontal lobotomy. Only one had a bimedial section of the pathways and only two had had a transorbital operation. There was no appreciable difference in these three and the remainder of the group. Six patients had had two operations. The second operation was carried farther back than the first and there can be no question that the second operation quieted the patient's violent behavior and made nursing care much easier, but it also produced more immediate evidence of intellectual deterioration. All six had convulsive seizures following lobotomy, that were more directly related to the second operation.

Convulsions following lobotomy were reported by Freeman and Watts[4] as "most embarrassing" in 12% of their patients, and by others with similar percentages. In this group of 83 patients there were 44 or 53% who had seizures at some time following lobotomy. For the most part these seizures occurred within the first year, but in a few cases, seizures occurred up to 20 years after the operation and the relationship to lobotomy was somewhat obscure, as there were no definitely localized abnormal findings in the EEG and there were no localized neurologic signs. This is in agreement with others.[7] In 1957[8] and again in 1960,[9] it was determined that in deep sleep only was there an asyncronous frontal slow wave activity in the EEG, apparently present in all lobotomized patients, irrespective of seizures. The seizures present in 44 of the 83 patients have been easily controlled with anticonvulsive drugs. It was suggested by Partridge[3] that more seizures occurred post-lobotomy in patients who had had electric shock treatment, prior to lobotomy. In this series of 83 patients, 44 or 53% had seizures post-lobotomy. Seventy-one of the 83 had had ECT prior to lobotomy. Fifty-four of the 83 had had insulin shock treatment. Fifty-three had had both ECT and insulin shock treatment prior to lobotomy. Of these 53 patients, 25 had no seizures and 28 had seizures. Only one had insulin alone and this patient had no seizure. Three patients had had neither insulin nor ECT treatment but one of these had a seizure post-lobotomy. Twelve patients who had ECT only, had no seizures and 13 with ECT only had seizures post-lobotomy. It is also curious that two patients with heavy ECT (200 treatments) and heavy insulin (160) treatments prior to lobotomy that neither had seizures post-lobotomy. I assume we cannot place all the blame for seizures on any single factor. The incidence of seizures is high in this group and the only common denominator is the frontal lobotomy operation. Some seizures occurred as long as 20 years after their lobotomy. This brings to mind personal experience with post-traumatic seizures occurring for the first time hours to 20 years after serious head injury. A relationship is assumed to exist between the trauma and the seizure but without definite proof of a direct relationship in that group of patients who had late occurring seizures. We can conclude in this group of lobotomized patients that the occurrence of seizures is not now a major problem, as in this group seizures were easily controlled.

Increased appetite with bulimia or polyphasia and gain in weight has been reported following lobotomy, but in this series of 83 patients, only nine are now obese and of these, three are diabetic. These three had no complications of diabetes and in spite of the fact that they could not be maintained on a diet, none of them had developed acidosis

or diabetic coma and none had visual, kidney or vascular complications, and none had neurologic complications at this time.

The tranquilizing drugs do make for better long-term management in these chronic schizophrenic disorders, although there are certain disadvantages namely the production of a Parkinson-like syndrome. In this group of 83 patients there are now 41 or 48% who show evidence of basal ganglia disorder. Although these Parkinson-like symptoms are most distressing to the nonpsychotic and even to the chronic schizophrenic, they are tolerated by most post-lobotomy patients without complaint. In fact in this lobotomized group generally, patients do not complain of pain and in a few instances patients have had fractures, coronary occlusion and perforated ulcer without complaining of pain. The ward nurses and attendants, as well as the ward physicians, must be alert to changes in behavior of these patients, as it may indicate the development of serious physical disorders.

It is generally agreed that lobotomy may still have a place in the treatment of the severe, completely disabling anxiety, phobic, obsessive disorders, and also that in certain selected instances it may be used in patients who have intractable pain syndromes.

In summary, this group of 83 patients who are the poor end results of lobotomy, there was immediate evidence of mild to moderate lessening of symptoms of restlessness and violence, although this improvement was not well sustained. Convulsive seizures although present in 53% of these cases is not considered a serious complication as seizures occurred mainly as single attacks and were otherwise easily controlled. The most important feature was that the schizophrenic disorder was not appreciably altered by the operation except to possibly hasten the process of emotional flattening and deterioration which at this stage, 20 years after lobotomy, is found to be both an emotional flattening and an intellectual deterioration of a mild to severe type. The diagnosis of chronic brain syndrome has had to be added to the prior diagnosis of schizophrenia. Present-day treatment with the tranquilizing drugs is more effective in quieting the patient than was lobotomy, but it does not alter the course of the psychosis appreciably, and in this group of patients the Parkinson-like symptoms arising from this form of treatment leave much to be desired.

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More detailed professional information available on request.

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Dosage and Administration: In women: one 250-mg. oral tablet three times daily for ten days. A vaginal insert of 500 mg. is available for local therapy when desired. When used, one vaginal insert should be placed high in the vaginal vault each day for ten days; concurrently two oral tablets should be taken daily.

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SOURCE: DUNCAN, G.G.: SCIENCE NEWS LETTER, 83:403 (JUNE 29) 1963.

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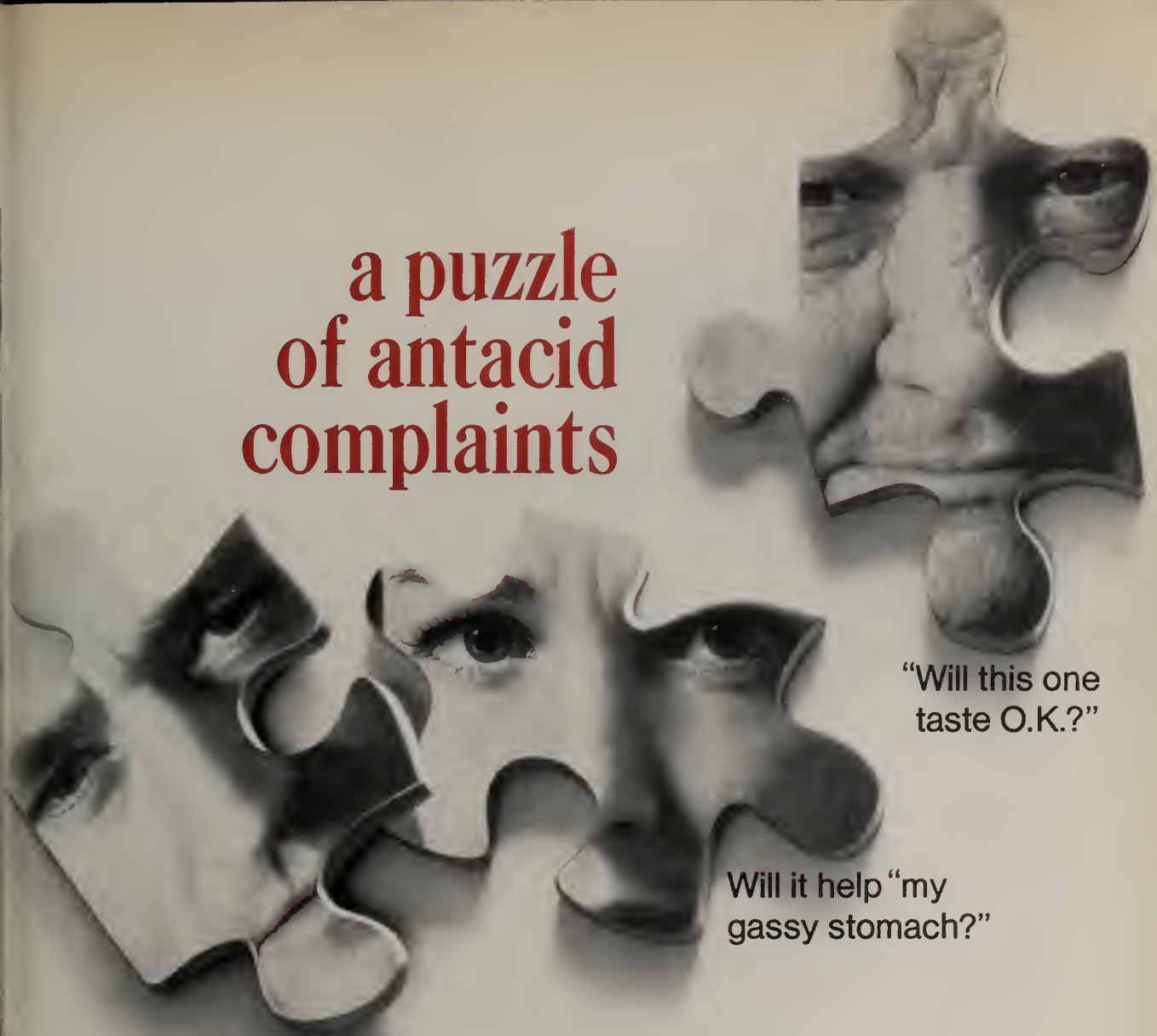
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68-11

Diagnostic Laboratory Medicine....

M. J. ALLISON, Ph.D.
E. GERSZTEN, M.D.
H. P. DALTON, Ph.D.
M. R. ESCOBAR, Ph.D.

Pulmonary Mycoses in the Richmond Area 1957-1966

A review of the hospital records of the Medical College of Virginia for the years 1957-1966 disclosed 78 patients with pulmonary mycotic disease, or 2.2 cases per 10,000 admissions. Eleven of these 78 patients had additional extrapulmonary lesions.

The cases are divided according to etiological agent, sex, race, and age as shown in Table I. It may be noted that these infections are most common in white males over the age of 41. *H. capsulatum* was responsible for 50% of the disease in these patients. It was followed by *N. asteroides* (14%) and *Aspergillus sp.* (11.5%). It is

TABLE I

DISTRIBUTION ACCORDING TO SEX, RACE AND AGE
1957-1966

INFECTION	SEX		RACE		AGE GROUP		
	M	F	W	C	0-20	21-40	> 41
Histoplasmosis	20	19	31	8	4	14	21
Nocardiosis	8	3	2	9	1	4	6
Aspergillosis	8	1	5	4	3	3	3
Blastomycosis	6	1	5	2	2	1	4
Cryptococcosis	4	2	2	4	—	1	5
Candidiasis	1	1	2	—	1	—	1
Actinomycosis	2	—	—	2	—	1	1
Phycomycosis	1	—	1	—	—	—	1
Candidiasis and Nocardiosis	1	—	1	—	—	—	1
Total	51	27	49	29	11	24	43

TABLE II

PRIMARY AND SECONDARY MYCOTIC DISEASE
1957-1966

INFECTION	PRIMARY		SECONDARY		
	Lung	Generalized	Lung	Generalized	Total
Histoplasmosis	31	3	5	—	39
Nocardiosis	2	—	8	1	11
Aspergillosis	—	1	6	2	9
Blastomycosis	4	1	2	—	7
Cryptococcosis	—	1	4	1	6
Candidiasis	—	—	2	—	2
Actinomycosis	2	—	—	—	2
Phycomycosis	—	—	—	1	1
Candidiasis and Nocardiosis	—	—	1	—	1
Total	39	6	28	5	78

interesting to observe that mixed infections occur in mycosis as in most other types of infections; there was one patient with *C. albicans* and *N. asteroides*.

The patients are classified in Table II on the basis of whether their disease was primary or secondary to some other condition. The most common primary and secondary lung diseases were histoplasmosis (31 cases) and nocardiosis (eight cases), respectively.

The conditions associated with secondary mycotic disease are listed in Table III. This table demonstrates that the majority of patients with pulmonary mycosis does not necessarily have a detectable predisposing associated condition as a prerequisite for the mycotic infection to develop. When these associated conditions are present, superimposition of the fungus disease on a previous bacterial infection, primarily tuberculosis, is by far the most common. This is followed in frequency by superimposition on a neoplasma, mainly lymphomas. Cases associated with diabetes mellitus are under 10%.

Table IV illustrates the method of diagnosis. Over 50% were diagnosed by tissue section either from biopsy or autopsy. The low frequency of isolation of the etiological agent may reflect neglect on the part of the clinician to use this procedure. It must be noted that the diagnosis of pulmonary mycosis on the basis of immunological tests is usually presumptive in nature. A clinical diagnosis without confirmatory tests could only be considered suggestive of this type of disease.

The outcome of the disease in this series of patients is shown in Table V. Mycosis

TABLE III

SECONDARY PULMONARY MYCOSES
ASSOCIATED CONDITIONS

INFECTION	Bacterial Infection	Diabetes Mellitus	Neoplasia	Renal Trans-plantation	Miscel-laneous	Total
Histoplasmosis	1	1	2	—	1	5
Nocardiosis	7	—	—	1	1	9
Aspergillosis	3	—	3	2	—	8
Blastomycosis	—	1	—	—	1	2
Cryptococcosis	2	1	1	—	1	5
Candidiasis	—	—	1	1	—	2
Phycomycosis	1	—	—	—	—	1
Candidiasis and Nocardiosis	—	—	—	—	1	1
Total	14	3	7	4	5	33

TABLE IV

PULMONARY MYCOSES
METHODS OF DIAGNOSIS

INFECTION	Tissue	Culture	Clinical	Immuno-logical	Total
Histoplasmosis	24	4	3	8	39
Nocardiosis	—	11	—	—	11
Aspergillosis	7	2	—	—	9
Blastomycosis	6	1	—	—	7
Cryptococcosis	6	—	—	—	6
Candidiasis	1	1	—	—	2
Actinomycosis	—	2	—	—	2
Phycomycosis	1	—	—	—	1
Candidiasis and Nocardiosis	—	1	—	—	1
Total	45	22	3	8	78

was the cause of death only in nine cases (11.5%). In the 33 patients in whom the mycosis was secondary to another disease, the death rate was 53%, but only 9% died of their fungal infection, the rest died of other causes. It may be noted that in this series the mortality rate was essentially the same for patients with primary and secondary mycosis.

TABLE V

PULMONARY MYCOSES
1957-1966

OUTCOME

INFECTION	PRIMARY				SECONDARY			
	Improved	Died		Total	Improved	Died		Total
		Fungal	Other			Fungal	Other	
Histoplasmosis	32	2	—	34	4	—	1	5
Nocardiosis	1	1	—	2	7	1	1	9
Aspergillosis	—	1	—	1	3	—	5	8
Blastomycosis	4	1	—	5	—	1	1	2
Cryptococcosis	—	1	—	1	—	—	5	5
Candidiasis	—	—	—	—	—	—	2	2
Actinomycosis	2	—	—	2	—	—	—	—
Phycomycosis	—	—	—	—	—	1	—	1
Candidiasis and Nocardiosis	—	—	—	—	1	—	—	1
Total	39	6	—	45	15	3	15	33

Division of Clinical Pathology
Medical College of Virginia
Richmond, Virginia

Study of Behcet's Syndrome

The cooperation of physicians is requested in the referral of patients for a continuing study of Behcet's syndrome being conducted by the National Institute of Dental Research at the Clinical Center, National Institutes of Health, Bethesda, Maryland.

Needed are patients with oral ulcerative lesions and other characteristic manifestations, such as arthritis, ophthalmologic inflammation, erythema nodosum, erythema multiforme, brain stem, meningomyelitic or organic confusional syndromes, or significant gastro-intestinal disease. Studies will include blood tests, electroencephalography and lumbar puncture.

Selected patients will be admitted to the Clinical Center for a period of two weeks or more. Upon completion of their studies, patients will be returned to the care of the referring physician who will receive a summary of findings.

Physicians interested in having their patients considered for admission to this study may write or telephone: Norman A. Cummings, M.D., Clinical Center, Room 2-B-10, National Institutes of Health, Bethesda, Maryland 20014. Telephone: 656-4000, Ext. 1363 (Area Code 301)

The Medical Society of Virginia . . . .

Baltimore: A Conference on Aging and Long Term Care—1967

About 600 people interested in aging and long term care met at the Emerson Hotel, November second and third, in a conference sponsored by the American Medical Association in cooperation with the State Medical Associations of Delaware, Maryland, New Jersey, New York, Pennsylvania, Virginia, West Virginia and the District of Columbia. I was privileged to represent The Medical Society of Virginia.

In his opening remarks Senator Walter F. Mondale noted that in our fast growing economy retirement at age thirty-eight was possible and may become practical. Americans, he said, traditionally feel guilty about leisure, as work is the guiding motivation of our culture, but this may have to change. It costs the country fifty seven and a half billion dollars annually for chronic illness and he felt that multiphasic screening, while expensive, might save money in time.

Dr. Schwartz, Chairman of the AMA's Committee on Aging, reiterated the Association's stand that geriatrics not be made a separate speciality, as the problems of the aged and aging cut across all of the fields of medicine, but Dr. Herman Seidel of the Maryland Commission on Aging disagreed. He recounted that the speciality of pediatrics went through a period of unacceptibility as a speciality until it was finally realized by the profession as dealing more than with the "little man". Older people desire a physician who is interested in their particular problems. This is really the first official voice to make a proposal for the geriatrician.

In a panel entitled Meeting the Challenge, Mr. Charles Barrows, nutritionist, brought out some interesting facts on the aging process. He stated that there is a decrement with age of the metabolic rate, glucose tolerance, cardiac output and vital capacity. There is cellular loss of muscle and nerve tissue, most marked in the highly differentiated cells. There is increasing evidence that aging is built into genes and that each cell is programmed through the D.N.A. and R.N.A. molecules, but that life cycles may be altered in simple animals like the rotofor, so that its egg laying may be altered, as to time, but that the total number of eggs laid in a life time remains the same.

Dr. Butler, a psychiatrist, stated that four per cent of those over sixty-five now live in institutions. He stated that circulation in the brain is unaltered unless impeded by arteriosclerosis, and that should this disease be eliminated, we should live to the age of one hundred, which would result in 65,000,000 people past the age of sixty-five by the year 2000. He felt that the breakthrough on arteriosclerosis was a definite possibility.

Dr. Sensabaugh, an educator, felt that even children should receive education for aging in the school and that education of older people should be selective beginning with literacy and progressing up the educational ladder through vocational education, socio economic education on up to retirement. He felt that educational television has great potential.

On health manpower Dr. Joseph Hamburg of Lexington, Kentucky, reported that in 1956 sixteen per cent of physicians in this country were foreign-trained, but by 1966 there were twenty-six per cent, fifty per cent of which came from underdeveloped countries whose need for physicians is pathetic. Last year of the 5,000 physicians added to the profession twenty-eight per cent were graduates of foreign medical schools. He commented that instead of importing physicians we should be exporting them in view of world needs. In commenting on nurses, he felt that the pay of the nurse is too low, that the utilization is inequitable, and there

is not enough career mobility. There are over 500,000 nurses in the country who are not working in the profession, and that if these could be encouraged to return to their profession even in part time service it would do much to relieve the shortage. Geographical maldistribution is also a contributing factor to our present nurse shortage.

Dr. Norbert Roberts, Associate Director of the Standard Oil Company of New Jersey, spoke on automatic multiphasic screening and observed that this is not practical for the entire population because of its enormous cost, and because of the paucity of data which would justify it. He admitted that a great proportion of chronic disease might be discovered but not necessarily cured as for instance, diabetes, stroke and coronary disease. It might eliminate certain risk factors in the production of disease but how much morbidity could be prevented is problematical.

In again discussing manpower, Dr. Hamburg denied that crisis is the proper description of our dilemma as statistics are not reliable. There has been no substantial change in the patient-physician ratio in spite of the population explosion. There has been a 25 to 30 per cent greater utilization of the physician's services than thirty years ago and a greater availability of medical care. To relieve the problem he suggested increase in group practice, more computer monitoring, as in coronary care units, less sophisticated help in hospitals for more of the nurses' work load, more flexibility in changing of the roles of health personnel, more accurate evaluation of health needs and a redistribution of care in a new package.

One of the most stimulating papers of the Conference was given by Dr. George Sharpe of Maryland on the work of a Medicare Coordinating Committee of the Montgomery Medical Society of Maryland. This Committee interested itself in the flow of patients from the hospital to extended care facilities to home, nursing homes or chronic disease hospitals. Four hospitals with 840 beds, ten nursing homes acceptable to Medicare with 900 beds were included in the study. Seventy one physicians participated on a rotating basis on utilization of the ten nursing homes. They set up criteria for the evaluation of each nursing home patient and were paid $25 an hour for their work from a government grant set up for the purpose. They asked themselves the following questions:

1. Why was the patient originally placed in the extended care facility?
2. To what extent can the patient be rehabilitated by being in the extended care facility?
3. How much and what kind of nursing care will be needed?
4. Would the patient be able to be cared for in the custodial type of nursing home?
5. What was the condition of the patient on admission to the extended care facility?
6. Has the physician seen and recorded the patient's condition, progress, and prognosis in the recent past?
7. Is there evidence of progress in the rehabilitation of the patient?

He concluded from the study that (1) A community-based utilization review program can give rise to a structure within the dynamic processes of patient care so that various community needs, such as home health care, can be determined and met. This could conceivably lead, for instance, to the use of community resources such as high schools, junior colleges and hospitals for the training of para-medical personnel. (2) The mobilization of personnel and resources in the community could be a significant factor in decreasing the costs of medical care. Not only could patients be placed in lower cost care situations but the need of physicians' time and involvement could be reduced. (3) A coordinating committee financed through intermediaries and under the wing of a medical society could

result in the following: Give rise to very objective utilization review, achieve area-wide standardization in utilization review, and keep the control of the flow of patients in the hands of physicians which is not only required by law, but is where it fundamentally should be. Traditionally this has been the role of organized medicine.

The concluding address entitled Our Common Dilemma—Retirement was given by Dr. Irving S. Wright, immediate past-president of the American College of Physicians. There are now 20,000,000 people past the age of 65, and by the year 2000 the number will be 65,000,000 when one hundred years may become the average life expectancy. Today 20,000 people are being kept alive by cardiac pace-makers. He spoke against the Bismarkian concept of compulsory retirement. This has become a part of American life because of a rigidity of both management and union on the grounds that the older worker stands in the way of the progression of the younger. The automatic way of retirement makes for simplification of the entire process including the development and implementation of pension plans. However, absenteeism is lower among those over 65 than among younger workers. He stated that the penalizing of those over 65 by confiscating the earnings above $1,500 was patently an unfair and ridiculous policy of the social security system. He urged Congress to abolish this feature of the Social Security Act and permit all workers to earn whatever they could.

He recommended that methods be developed to screen out those unable to work effectively after 65 years of age from those who could, as biological age is not necessarily related to chronological age. Much talent of the oldsters is being lost to society as is demonstrated by the foundation of independent retired tool and dye workers who have set up their own shop for contract work which has proven highly successful. Other top-flight elders have set up consultative services made up of retired executives to think and advise for industry for a fee, pooling their broad experience in a worthwhile venture not only for themselves but for industry and management.

It was your reporter's privilege to have attended a similar conference ten years ago and in going over the report of that conference, I find we are dealing with almost exactly the same problems then as now. The only difference seems to be that the problems have intensified and the cost of health care has doubled. There does seem to be a more widespread understanding of the problems and a more widespread base of responsibility which now includes the Federal Government but we do not seem to be approaching any more satisfactory solution than in 1957. As a matter of fact, we seem to be more confused about how we are going to move and in what direction. Obviously, the Great Society has no intelligent solution. We must somehow go back to the fundamentals upon which our country was founded and cease liberal experimentation at the exepnse of national solvency and national sanity.

JOHN P. LYNCH, M.D.

Medicine and Religion

The Southeastern Medicine and Religion Regional Meeting was held at The Hilton Inn, Atlanta, Georgia, March 16, 1968.

This meeting included West Virginia, Virginia, North Carolina, South Carolina, Georgia, Florida, Mississippi, Alabama, and Louisiana, all of which were represented except West Virginia.

The meeting began with a Continental Breakfast. Dr. Paul B. McCleave, Director of the Department of Medicine and Religion of the American Medical Association, gave the Invocation.

Mr. Arne Larson, Assistant Director of the Department of Medicine and Religion of A.M.A., gave a discussion on Organizational Planning, touching on county medical societies, the selection and appointment of chairmen and committee members and

methods for the appointment of proper members, giving examples of programs at the University of Iowa and Kansas. He stressed the cost of printing and mailing material and assured us that all such material was available on request. He stressed the point that before state meetings are scheduled, the state chairman should be certain that such meeting not conflict with other meetings.

He also suggested that at our annual state meeting, or at the meeting of the House of Delegates, some time on the program should be allotted to an A.M.A. Representative of Medicine and Religion to present, in brief, the program of M & R in an attempt to impress upon the component state chapter, or delegates, the importance of having an active Medicine and Religion Committee in each component chapter.

He also urged that the program chairman of each state chapter schedule a showing of the film "The One Who Heals" and also recommended that this film be shown at all State Officer Training Programs.

Mr. Robert L. Coons, of the A.M.A. office of Medicine and Religion, gave an enlightening discussion on State Workshops for county chairmen.

Dr. N. B. Baroody, Jr., South Carolina, gave a breakdown on State Workshop Experience. His remarks were especially gratifying and pleasing to me since all of his suggestions have been in operation in Virginia for the past 12-18 months.

Dr. Paul B. McCleave then discussed Resources for help in which he reviewed the Program Guide, stressed the importance of showing the film "The One Who Heals." He then presented an informative discussion on Medical Schools and Seminary Programs.

After a very delicious luncheon, we divided into two groups, one moderated by Mr. Larson and the other by Mr. Coons. At these meetings, we considered questions from the members, which were answered by the moderator or members, individual state planning for 1968-1969, and a review of resource material.

Dr. McCleave spent time with each group answering questions and making suggestions.

The afternoon session concluded with a Benediction by Dr. James A. Long, Jr., Florida, a member of the A.M.A. Board, who is Chairman of the Committee of Medicine and Religion of A.M.A.

JOHN WYATT DAVIS, JR., M.D.
Acting Chairman from Virginia

Let's Reminisce!

COOKING BY ELECTRICITY.—A curious experiment (says the Courier de Paris) took place on Monday at St. Denis, at the residence of M. Gisquet, formerly prefect of police at the commencement of the reign of Louis Phillippe, and now oil refiner. By means of an apparatus invented by M. Bullot, and in which electricity is the only agent, six pounds of beef were perfectly cooked in five minutes and ten seconds. Several trustworthy witnesses, watch in hand, verified this rapid act of cookery. At the end of the 310 seconds the boiled meat and the broth made from it were tasted, and both were found to be excellent, although a little too salt—a fault, however, not attributable to the apparatus. (Virginia Medical Journal, September 1857)

Woman's Auxiliary....



Entire group from Medical College of Virginia. *Back row,* left to right: Floyd Atkinson, sophomore; Edward Lilly, junior, and president of SAMA chapter; Mallory Read, freshman; John Reynolds, junior. *Second row:* Linda Lilly, Kay Read, and Jane Reynolds. *Front row:* Robin Kappler and Marlene Meyer.

Richmond Chapter of WASAMA Is Largest Chapter in U.S.

Announcement of the Sally Tompkins Chapter of The Woman's Auxiliary to the Student American Medical Association as the largest chapter in the country, was made in Atlanta at the Region I convention, February 16th. Mrs. G. Edward Kappler, President of the Richmond Chapter, headed the delegation. Others representing the chapter were Mrs. Mallory Read, Mrs. Edward Lilly, Mrs. Julian Meyer and Mrs. John Reynolds.

The Sally Tompkins Chapter was formed in August through the efforts of the Women's Auxiliary to The Medical Society of Virginia and the Women's Auxiliary to the Richmond Academy of Medicine. Girls who were leaders in other groups were called upon to plan the association and officers were appointed.

In October the chapter held its first meeting—a fashion show given by a local women's store with modeling done by our members. It was highly successful and the girls showed their approval by signing membership cards on the spot. One hundred and three charter members signed in at the Valentine Museum, a lovely historic place, where the first meeting was held. Wine sherbet punch and petits fours were served by our hostesses, the Women's Auxiliary to the Richmond Academy of Medicine.

In November the Executive Committee met to plan the meetings that would take place this year and the following programs were decided upon:

December: "So You Wanna Be A Doctor's Wife?" featuring a panel of local physicians' wives discussing the problems and joys of being a doctor's wife, followed by a question and answer period.

February: "Pitfalls into the Valley of the Dolls—Drugs and the Doctor's Wife" presented by a panel of physicians—one in charge of the pharmacology department, one a psychiatrist, and one just plain good looking!

April: "Types of Practices—Pros and Cons" with a panel of physicians and a question and answer period. Husbands were invited.

June: Elections and card party at a local hotel with prizes donated by businessmen.

We had a children's Christmas Party for our own children. A local story teller told a Christmas story, and one member who has her own TV music program for children entertained for half an hour. Cookies were served and punch too. The children loved it! One member's Daddy came as Santa and gave apples to all the children.

We had a spaghetti dinner for 150 people to raise money and did we raise money! The food was donated through a friend and we made $187.80 on it. The people who came to dinner got spaghetti, salad, garlic bread, dessert and a coke—all for $1.00. They raved about it! Where did we have it? At Medical College of Virginia, and

served it to hungry interns, residents, nurses, etc.

We are planning a Doctors' Coffee Break for Doctors' Day and will serve free coffee and cake to residents, interns, attendants, and students.

Besides all of our other activities, we sold five hundred stuffed toys, that we bought for 60¢ each, for $1.00 each. And we are working on a cookbook.

Our president and other delegates attended state convention as guests and learned a great deal there—also met Mrs. James L. Rothermel, President-elect of National WASAMA and Mrs. Paul Rauschenback, WA-AMA National Liaison to WASAMA. Each month our president attends the meetings of WARAM as their guest.

As a community service project, we have selected an easy one. Admission to our next meeting will be one or more articles of used clothing—Mommy's, Daddy's and Child's. This will be sorted and donated to a public school where there is a high incidence of dropouts due to improper clothing. They will mostly go to a high school here in a very poor section of town.

Another service project in the frying pan is working as volunteers in the emergency room to calm families of patients. And our ER is a frying pan!

We give lovely favors at each meeting. At the first meeting we distributed Chap-Ans Hand Cream. Mrs. Hoge, our sponsor, got us perfume that is $6.50 a bottle, as an advertisement for Montaldos. We are giving bath oil and hand lotion made by a pharmaceutical house in Virginia at our next meeting.

We have published a monthly newsletter, *The Nucleus,* and have reached all our members by phone before each meeting. We have had exceptional luck with publicity.

At our next meeting, we are going to raffle off a dish, donated by Mrs. Hoge, at 25¢ a chance.

We have interest groups which meet once a month in cooking, sewing, book review, knitting, slimnastics, bowling, children's play group and bridge.

To date our paid membership is 160.

Robin Kappler (Mrs. G. Edward)

Dateline San Francisco—AMA Auxiliary to Hold 45th Annual Convention— Conducted tours through a unique rehabilitation mobile unit and an afternoon of continuous health education films will be new features at the 45th Annual Convention of the Woman's Auxiliary to the American Medical Association. Headquarters for the June 16-20 meeting will be the St. Francis Hotel, San Francisco, California.

Highlighting the four-day session will be the new mobile demonstration unit called "Homemaking Unlimited". This five-ton coach, sponsored by the School of Home Economics of the University of Nebraska, shows handicapped housewives how they can be self-reliant homemakers despite physical limitations. During the convention, the unit will be displayed on Powell Street, across from the St. Francis Hotel.

On Sunday, June 16, the Auxiliary will hold a reception for the president, Mrs. Karl F. Ritter, Lima, Ohio, and president-elect, Mrs. C. C. Long, Ozark, Arkansas.

The convention will formally open at 9 a.m., June 17. Mr. Philip Lesly, president of the Philip Lesly Company, a Chicago-based public relations firm, and counsel on PR and communications to the AMA, will deliver the keynote address.

Monday luncheon will honor the leaders of women's volunteer organizations throughout the United States. Featured speaker will be Henrik L. Blum, M.D., Clinical Professor of Community Health Planning, University of California School of Public Health. Dr. Blum will discuss "Program Planning and Implementation".

Milford O. Rouse, M.D., AMA President, will be the guest speaker at the June 18th luncheon honoring national Auxiliary past presidents and AMA Officers, Trustees and wives. At this time Mrs. Ritter will present the Auxiliary's contribution to the Ameri-

can Medical Association Education and Research Foundation. Last year's gift totaled $384,649.

The afternoon meeting will feature a program by the Communications Committee relating to state publications. Following this program, films, including several on sex education, drug abuse, physical fitness and nutrition, will be shown. This new feature will provide Auxiliary members with a catalog of available health education materials to be used in conjunction with existing Auxiliary programs.

The postconvention conference, usually scheduled for Thursday morning, will be held on Wednesday afternoon to give Auxiliary members free time on Thursday to visit areas of San Francisco. The Board of Directors will meet Thursday morning, instead of Wednesday afternoon.

The Auxiliary will also sponsor a program of daily activities for the pre-teens and teenagers of Auxiliary members and guests. Lunch in Chinatown and a cable car ride are among the events being considered.

Norfolk Auxiliary Conveys Honorary Membership to Two—Mrs. Theodore Adler, President of the Norfolk Auxiliary, gave Honorary Membership in February to two past State presidents. Her presentation follows:

> "It is a rare privilege I have today to honor two members of our auxiliary. Both of these women are outstanding as they have contributed to all phases of community life and has each been president of the Norfolk County Auxiliary and the Woman's Auxiliary to The Medical Society of Virginia.
>
> Mrs. Kalford W. Howard has lived in Portsmouth all of her life. In addition to being president of the Norfolk and Virginia Auxiliaries, she has served in this capacity for the Southern Medical Auxiliary.
>
> One of the first two women to serve on the Portsmouth General Hospital Board, she also served as president of that hospital auxiliary. However her activities have not been limited to the medical profession. Mrs. Howard has served as president of the PTA, has been active in the Portsmouth Community Concert Association, the Portsmouth Chapter of the D.A.R., Portsmouth Women's Club and in Monumental Methodist Church. In 1957, Mrs. Howard was named Portsmouth's Woman of the Year.
>
> The accomplishments of Mrs. John Randolph St.George are equally impressive. Raised in Portsmouth in the atmosphere of the medical profession, Louise Waller St.George has not been a bystander. Besides being a former president of the Norfolk and Virginia Medical Auxiliaries, she has been active in the Woman's Auxiliary to the Virginia Academy of General Practice and a former president of the Auxiliary to the Kings' Daughters Hospital, which is now Portsmouth General. Other presidencies include the Elizabeth River Garden Club, Episcopal Church Women of St. John's Episcopal Church. Her interests and hobbies extend into community concerts, choir work, gardening and little theater.
>
> Mrs. Howard and Mrs. St.George helped to organize the Woman's Auxiliary to the Portsmouth Academy of Medicine. However, they have remained loyal members of the Norfolk Auxiliary. In recognition of your devotion and signal service, I am proud to present to each of you a certificate of Honorary Membership in the Woman's Auxiliary to the Norfolk County Medical Society."

Honors to Mrs. Michael Puzak were presented during the State President's luncheon, March 5th. Alexandria, Fairfax and Arlington Auxiliaries joined together in presenting Mrs. Puzak with an inscribed tray in appreciation of her outstanding leadership as well as her most successful chairmanship of the State Convention last October.

Editorial . . .

The Training of X-Ray Technicians

IN PREPARING A PAPER on the problems of the education and training of nurses, I became aware of a striking parallelism between their problems and those of the x-ray technician. Both professions, composed for the greatest part of dedicated, loyal and self-effacing people, could be traveling along similar, perilous paths.

Responsibility for his own filming in his training or in his early post-training period should have given the radiologist total familiarity with the technical principles of radiographic exposure and processing. Only he can set the contrast scale, for example, that meets his professional demands. He must be the guiding light, as another example, in setting up technique charts and routines of positioning, because he occupies the best place to evaluate such matters in light of his professional experience. Delegation by the radiologist of such tasks to others, regardless of their technical competence, is an abrogation of his professional responsibility.

Given such a radiologist practicing in a small department in a small community, a receptive pupil, and a satisfactory teaching environment, a potentially ideal teaching situation for common radiographic principles and techniques exists. Nothing that the pupil does is beyond the immediate inspection of her mentor. That she develops within the image of her tutor is acceptable since he is ultimately responsible for her future professional conduct as long as she remains in his employ. If the pupil cannot be entrusted with the emergency work after six weeks of such training, she is considered unsatisfactory and dismissed. She is paid a living wage from her first day in the department. The wage is increased rapidly in proportion as she increases in value to the department. Praise is lavish for things well done; blame is heavy for things poorly done.

This intimately controlled apprentice system has proved eminently satisfactory for our needs over the past 20 years. We had been forced into the system for two main reasons: 1. the difficulty in securing the traditionally trained technician for services in this small community with her social life even further limited by the presence of a large women's college; 2. the significant time required to indoctrinate the technician trained elsewhere in "our way of doing things", right or wrong though this way might have been. Any radiologist is invited to inspect our system in operation.

The present trend in official organizational circles is to abolish training of this type; even training in the hospital schools is beginning to receive auras of deprecation in favor of a move to the college campus with its baccalaureate programs. Shades of Nursing!

Such trends are bad: 1. Fewer will enter x-ray technology because of the higher cost of more involved or collegiate programs; 2. Fewer will enter because of the longer time required for such programs before the entrant can make a living; 3. Fewer technician-practice years will be

available to the community because of the longer preparation required; 4. A higher cost for technologists' services accrues to the community because technologists so prepared must expect a higher financial reward, despite lack of clear evidence to this writer that their day-to-day services are superior to those of the apprentice-trained; 5. Many technicians after prolonged training tend to abandon the workaday department to enter the greener allied fields such as representatives for film or equipment manufacturers; 6. Some young people who give promise of making satisfactory technicians because of certain natural qualities are dropped by the wayside of an overly rigorously oriented academic program. The sum of all these considerations, which are parallel to those of nursing, is fewer technicians at the time when the need is greatest.

Any consideration of x-ray technology as an independent profession is folderol. The radiologist is morally, legally, and professionally responsible for each and every official act of his technician, his *agent*. Since the radiologist is licensed, licensing his agent appears superfluous and illogical. Licensure of technicians can, and probably will be, so set up as to do away completely with this type of apprentice training, a serious encroachment upon the professional rights of the radiologist who chooses to train his technical help in his own way. Big government with its unprecedented rise of the administrative class, who usually know less than the people they administer, will make us all the same, as monotonously homogeneous as a row of "in" teen-agers.

The death knell for the apprentice-type training has possibly already been sounded though the sound has not quite yet reached us in the distant fields. Nevertheless we radiologists in the smaller communities that could support no formal training school, who would have a difficult time manning our little departments if we had to depend on technicians trained elsewhere, should make our voice heard for our right to train our own technicians, for whose every professional action we willingly admit moral, legal, and professional responsibility.

CHRISTIAN V. CIMMINO, M.D.

Lest We Forget—A Remarkable Virginian

"For when the One Great Scorer comes
To write against your name,
He marks not that you won or lost—
But how you played the game."

—GRANTLAND RICE

NEAR PETERSBURG, VIRGINIA, on 13/June/1786, he was born. In 1805, at the age of nineteen, he entered the College of William and Mary where he studied law.

In 1807 he went to live in Charleston, South Carolina, but as the clouds of the War of 1812 seemed to him to be gathering, he went to Washington where he offered his services and in 1808 he was commissioned as a Captain in the Artillery. Who was this Virginian?

He returned to Richmond and Petersburg where he recruited a company and was ordered with it to New Orleans. In 1812, as a Lieutenant Colonel, he was sent to the Niagara frontier and fought at Queenstown where, in 1813, he was taken prisoner. However, he was repatriated the same year and was promoted to Colonel. A year later he was promoted to Brigadier General to be followed within a few months by promotion to Major General. His exactness in dress and behavior won him the name "Fuss and Feathers." In July of 1814 he distinguished himself at the Battle of Chippewa and Lundy's Lane. He was twice wounded at Lundy's Lane. Who was he?

For fifty-three years, i.e., from 1808 to 1861, this remarkable man wore the uniform of the United States Army. During the last twenty years of this period he was Commanding General of his Nation's Army. Among the more difficult tasks performed or noteworthy expeditions led by him were the following:

1. In 1832 he led an expedition to the middle west where he negotiated treaties of peace with a number of Indian tribes including the Saux, the Fox, the Winnebago, the Sioux and the Menominee tribes. Who was this man?

2. He made a journey to South Carolina to observe the progress of the Nullification Movement.

3. In 1836 he led an expedition against the Seminole Indians in Florida.

4. In 1838 he had supervision of the removal of Cherokee Indians from Georgia, North Carolina, Alabama and Tennessee to the reservations west of the Mississippi that had been set aside for them by treaty.

5. Also in 1838 he went to the Niagara River to put an end to the acts of Canadian insurgents in violation of American neutrality.

6. In 1839 he headed a similar mission to Maine to restore tranquillity between the citizens of Maine and New Brunswick who were disputing the possession of land along the Aroostok River. Who was he?

7. In 1859 he made a journey to the northwest to adjust a dispute between American and British officials concerning the joint occupation of San Juan Island in Puget Sound.

8. It cannot be gainsaid, however, that his greatest achievement was the brilliant Mexican campaign in 1847. As senior officer of the U. S. Army, and with Ulysses S. Grant as a subaltern, he was in command of the invading expedition. He captured Vera Cruz and won victories at Cerro Gordo, Contreras-Churubusco, Molino del Rey and Chapultepec. His campaign was crowned by the capture of Mexico City on 14/September/1847. Who was this man? Sam Houston was a contemporary Virginia, but the individual in point was not Sam Houston.

In March 1848 he received from the U. S. Congress a vote of thanks and a gold medal struck in commemoration of his victories.

In 1852 he received the nomination for President of the United States. In this campaign he was backed by the Whigs who split on the Slavery question. He was accorded the electoral votes of only Kentucky, Virginia, Massachusetts and Vermont. Who was he?

In 1852 the brevet rank of Lieutenant General was created especially for him. Although a Virginian, at the outbreak of the Civil War he remained at the head of the United States Army and directed operations from Washington until 1861. While his age, 75, constituted the official reason for his relinquishment of his office as Chief of Staff of the Army, it was generally acknowledged that his aversion to directing a military force in opposition to his fellow Virginian, Robert E. Lee, was an added and significant stimulus to his retirement.

He visited Europe for a short stay and after his return, wrote his memoirs which were published in 1864. In 1865 he presented a gift to Ulysses S. Grant inscribed: "From the oldest to the greatest General."

At West Point, New York, on the 29th day of May 1866—one hundred and two years ago this year—two weeks and one day short of his eightieth birthday—he died, and was buried in the National Cemetery at West Point.

His first name was Winfield—General Winfield Scott.

In a superbly commendable manner, during an era when uncommon courage of conviction and an abiding faithfulness to his conscience and to his oath of office were *sine qua non* to his high order of loyalty to his country, he played the game of life. Of him it may well be reckoned, as Mark Antony observed with respect to Brutus: "The elements were so mixed in him that Nature might stand up and say to all the world, 'this was a man!' " Or, if one might be permitted to paraphrase a few lines from Parkenham Beatty's "To Thine Own Self Be True", it could be said that General Winfield Scott, by his own soul's law, learned to live and hoped his hope and prayed his prayer. He fixed on future's goal his face and neither tarrying by the way, nor letting his feet be lured to stray, he played out his own appointed part. So that, at last, from his lofty station on the side of his mountain he could look back over the course of his long day's journey with that satisfaction that singularly can come, not from a knowledge of merely having achieved success, but, from the realization moreover of having deserved it.

H. Lamont Pugh, M.D.

News . . . .

Calendar of Events

Virginia Academy of General Practice—Annual Scientific Assembly—The Chamberlin—Fort Monroe—May 9-12, 1968.

Medical College of Virginia Alumni Scientific Assembly—Richmond—May 31, 1968.

American Medical Association—Annual Meeting—San Francisco, California—June 16-20, 1968.

Seaboard Medical Association—Annual Meeting—Nags Head, North Carolina——June 21-23, 1968.

Medical Aspects of Sports—Sponsored jointly by The Medical Society of Virginia and Virginia High School League—University Hall—Charlottesville—July 21, 1968.

Health Executives Workshop—Sponsored by Virginia Council on Health and Medical Care—Hotel Roanoke—August 28-20, 1968.

Annual Cardiovascular Symposium—Sponsored by Tidewater Heart Association and Council on Clinical Cardiology, American Heart Association—Lake Wright Motel—Virginia Beach—September 6-7, 1968.

The Medical Society of Virginia—Annual Meeting—Hotel Roanoke—October 13-16, 1968.

New Members.

The following new members were received into The Medical Society of Virginia during the month of March:

John Fergus Alksne, M.D., Richmond
Douglas Kenley Armbrister, M.D., Marion
Mary Williamson Bartone, M.D., Fairfax
Charles George Bratenahl, M.D., Fairfax
John Mills Britt, Jr., M.D., Franklin
Jerry Brice Crum, M.D., Roanoke
Behrooz Dayanim, M.D., Portsmouth
Andrew Ivo Dobranski, M.D., Washington, D. C.
Harold Leopold Floyd, M.D., Richmond
David Garrison Fluharty, Jr., M.D., Newport News
Lyman Avard Fulton, M.D., Suffolk
Jose Antonio Gutierrez, M.D., Falls Church
William Wallace Harris, Jr., M.D., Covington
Howard Courtenay Harrison, M.D., Virginia Beach
Hooshang Hooshmand, M.D., Richmond
Eileen El-Dorado Johnson, M.D., Lynchburg
Alva Rex Kirkley, M.D., McLean
Jerry Lee Miller, M.D., Nickelsville
Niels Henrik Oster, M.D., Tappahannock
Robert Edward Rider, M.D., Martinsville
Edward Underwood Scherer, Jr., M.D., Clifton Forge
Ahmad Shoabi, M.D., Norfolk
Edward Guerrant Thompson, M.D., Winchester
Yonne Dorothy Varese, M.D., Newbern
David Bruce Young, M.D., Norfolk

Dr. William H. Barney

Has been appointed to the board of directors of the Lynchburg General Hospital. He is the first physician to serve in this capacity.

Dr. James V. Jordan,

Covington, was recently honored at a dinner party at The Homestead in recognition of his long service to the community. He has practiced in the Covington-Alleghany County area since 1920. He was presented with a gift of a humidor and fifty cigars.

Dr. A. Ray Dawson,

Richmond, has been appointed chairman of the department of physical medicine and rehabilitation of the Medical College of Virginia. He has been acting chairman since April 1967.

Dr. Thomas H. Iden,

Berryville, has been appointed to the advisory board to the board of directors of the Farmers and Merchants National Bank of Winchester.

Dr. James H. Masterson

Has been re-elected chief of the Medical Staff of the National Orthopaedic and Rehabilitation Hospital of Arlington.

Annual Scientific Assembly.

The Eleventh Annual Scientific Assembly, sponsored by the Alumni Association and the School of Medicine of the Medical College of Virginia, will be held in Porter Auditorium on May 31st. The program, which will begin at 9:30 A.M., is as follows: What's New in the School of Medicine by Dr. Kinloch Nelson; Management of Acute Respiratory Failure by Dr. James P. Baker; Management of Rheumatoid Arthritis by Dr. Elam C. Toone, Jr.; Skin Disorders of Interest to Clinicians by Dr. Kenneth W. Blaylock; Glucose Tolerance and Endometrial Carcinoma by Dr. Leo J. Dunn; and Transplantation by Dr. David M. Hume.

Medical Seminar Cruise.

Georgetown University School of Medicine is sponsoring a medical seminar cruise aboard the luxurious cruise ship, the Victoria, sailing from New York on November 6th on a 12-day Caribbean cruise. The program will consist of formal lectures, panels and discussions on a wide variety of subjects. There will be 24 hours of instructions.

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Obituary . . . .

Dr. James Obediah Fitzgerald, Jr.,

Richmond, died March 20th at the age of eighty-one. He received his medical degree from the former University College of Medicine, Richmond, in 1913. In addition to his practice, Dr. Fitzgerald was widely known for his photography. His works were displayed throughout the United States, Canada, South America, England and other parts of the world, and he had a one-man show of 60 of his prints at the Smithsonian Institution in Washington in 1945. Dr. Fitzgerald was a member of numerous camera and photographic societies, among them being a life and cornerstone member of the Photographic Society of America and a member of the Top Photographic Society, an honorary organization for the top photographers of the world. Dr. Fitzgerald's particular interest in medicine was in physical therapy and in using spectral rays his interest in photography was fostered.

Dr. Fitzgerald had been a member of The Medical Society of Virginia for fifty-three years and was a member of the Fifty Year Club.

His wife, a son and a daughter survive him.

Dr. Doris Cleo Boatwright,

Marion, died February 22nd. He was sixty-seven years of age and a graduate of the Medical College of Virginia in 1924. Dr. Boatwright established his practice in Marion in 1928 and practiced there until his retirement. He was a past president of the Smyth County Medical Society and had been a member of The Medical Society of Virginia for forty-one years.

In addition to his practice, Dr. Boatwright began at an early age to collect Indian relics and his interest in history and antiques continued throughout his lifetime. In later years he took up painting and some of his canvasses were exhibited in local shows.

His wife and a son survive him.

Dr. Folkes.

Clifford A. Folkes was born in Richmond in 1892. After graduating from Richmond College (now the University of Richmond), he entered the Medical College of Virginia and received his M.D. degree in 1915.

After pursuing a number of graduate courses in ophthalmology and otolaryngology, he limited his practice to these specialties for more than forty-eight years. Despite the demands of a large private practice, he found time to act as a specialist in his fields at the Virginia Penitentiary and was a Medical Consultant for the entire Virginia Penal System.

His membership in the following attests to his wide interests: Richmond Academy of Medicine, The Medical Society of Virginia, Richmond Eye, Nose and Throat Society, Virginia Society of Ophthalmology and Otolaryngology, founder member of the Caduceus Club and Elder of the Grace Covenant Presbyterian Church.

Four years before his death, a series of heart attacks precluded the practice of medicine, but he found idleness intolerable. Each day he could be found acting as a volunteer at the United Givers Fund. One of the greatest joys of his life occurred a few weeks before his death when he was awarded a plaque naming him as "The Most Valuable Volunteer Worker" of the year.

Though a specialist his interests were wide. He understood the human aspects of medicine and saw far beyond the purviews of the opthalmoscope and laryngoscope.

He was a skilled and understanding doctor in the eyes of his patients and a beloved physician and friend in the hearts of the unfortunates in the Penitentiary.

He is survived by his wife and his daughter.

THEREFORE BE IT RESOLVED that a copy of these resolutions be entered in the minutes of the Richmond Academy of Medicine and a copy sent to his family.

DOUGLAS G. CHAPMAN, M.D.
J. WARREN MONTAGUE, M.D.
EMANUEL U. WALLERSTEIN, M.D., *Chairman*

Dr. Weinstein.

Abraham Isaac Weinstein, a member of this Academy for fifty-three years, died in his seventy-sixth year, on November 4, 1967.

Dr. "A. I.", as he was affectionately known by his many friends, was born in Richmond on August 1, 1892. Following graduation from Richmond High School, later to be known as the John Marshall High School, he entered the Medical College of Virginia, from which he received, in 1913, the degree of Doctor of Medicine. He spent the next twelve months serving an internship at Richmond's City Home. The loss of an eye in an accident during this period necessitated his foregoing long laid plans for specialized training in orthopedic surgery.

Dr. "A. I.'s" favorite hobby was fishing in the Tidewater rivers of Virginia, though a busy practice prevented his indulging very often in this pastime. Then he enjoyed his rose garden, from which each morning, in season, he would carefully select and cut a rose to wear in his buttonhole. We have called these his favorite hobbies and, yet, it would probably be nearer the truth to say the care of his patients was his favorite hobby above all others. Fortuitous investments made early in his professional career obviated the necessity of his continuing active practice beyond the usual retirement age of sixty to sixty-five. But the care of his beloved patients was "life" itself to Dr. "A. I.". He was "on call" every minute of both day and night and with true dedication and innate kindness, he catered especially to those of advanced age.

At Christmas time he derived much pleasure from personally buying fruit and other delicacies which he would pack in baskets and deliver to his older, retired patients. Often he would tuck into them a piece of jewelry or some especially appropriate gift. He told us that the patients to whom he took these baskets had, in most instances, contributed generously to his welfare for services rendered over a period of thirty to fifty years, when they were gainfully employed, and now he felt that it was his turn to contribute to their welfare. How many he treated without thought of compensation no one will ever know. The number, however, must have been large judging from the many patients who told us of his numerous kindnesses.

Dr. "A. I.'s" cheerful manner was one of his most endearing traits. Certain quaint mannerisms and customs particularly endeared him to the nurses and other personnel of Stuart Circle Hospital. They looked forward each day to his approach, signaled by a low off-key whistle and holding in each hand a plastic bag of assorted candies which he would leave at the nurses' station while he visited patients. By the time he reached the first floor and had visited the girls in the record room and clinical laboratory, the candy bags were empty.

Not only was he generous with his time, but with contributions to many charitable organizations, his favorites being the Richmond Boys' Home and the Sheltering Arms Hospital. He also was liberal with his contributions to organizations of the Jewish faith, to which he was born, and to the Park View Baptist Church, which he joined in 1942.

Dr. Weinstein was a member of The Medical Society of Virginia from which, in 1963, he received a certificate for fifty years of active practice, and, to the American Medical Association. He was also a Mason, being a fifty year member of the Richmond Randolph Lodge, Number 19 A. F. and A. M. and Samis Grotto.

He is survived by his wife, a daughter and four sons.

THEREFORE, BE IT RESOLVED that a copy of these resolutions be placed in the minutes of the Richmond Academy of Medicine, a copy to be sent to the Virginia Medical Monthly and a copy to be presented to the family of Dr. A. I. Weinstein.

JAMES B. DALTON, JR., M.D.
RICHARD A. MICHAUX, M.D.
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Side effects include drowsiness; dizziness; nausea; excitement; hypotension; fine tremor; jitteriness; weakness; headache; heartburn; anorexia; increased perspiration; incoordination; allergic-type reactions manifested by skin rash, swelling of face and tongue, itching; numbness and tingling of limbs, including peripheral neuropathy; activation of schizophrenia which may require phenothiazine tranquilizer therapy; epileptiform seizures in chronic schizophrenics; temporary confusion, disturbed concentration or, rarely, transient visual hallucinations on high doses; evidence of anticholinergic activity, such as tachycardia, dryness of the mouth, blurring of vision, urinary retention, constipation; paralytic ileus; jaundice; agranulocytosis.

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COMPOSITION: Imferon (iron dextran injection) is a well-tolerated solution of iron dextran complex providing an equivalent of 50 mg. in each cc. The solution contains 0.9% sodium chloride and has a pH of 5.2-6.0. The 10 cc. vial contains 0.5% phenol as a preservative.

ADMINISTRATION AND DOSAGE: Dosage, based upon body weight and Gm. Hb/100 cc. of blood, ranges from 0.5 cc. in infants to 5.0 cc. in adults, daily, every other day, or weekly. Initial test doses are advisable. The total iron requirement for the individual patient is readily obtainable from the dosage chart in the package insert. Deep intramuscular injection in the upper outer quadrant of the buttock, using a Z-track technique (with displacement of the skin laterally prior to injection), insures absorption and will help avoid staining of the skin. A 2-inch needle is recommended for the adult of average size.

SIDE EFFECTS: Local and systemic side effects are few. Staining of the skin may occur. Excessive dosage, beyond the calculated need, may cause hemosiderosis. Although allergic or anaphylactoid reactions are not common, occasional severe reactions have been observed, including three fatal reactions which may have been due to Imferon (iron dextran injection). Urticaria, arthralgia, lymphadenopathy, nausea, headache and fever have occasionally been reported.

PRECAUTIONS: If sensitivity to test doses is manifested, the drug should not be given. Imferon (iron dextran injection) must be administered by deep intramuscular injection only. Inject only in the upper outer quadrant of the buttock, not in the arm or other exposed area.

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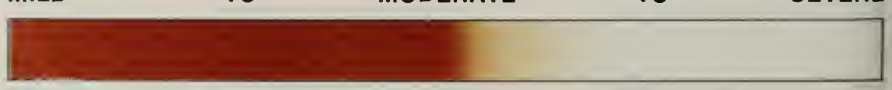
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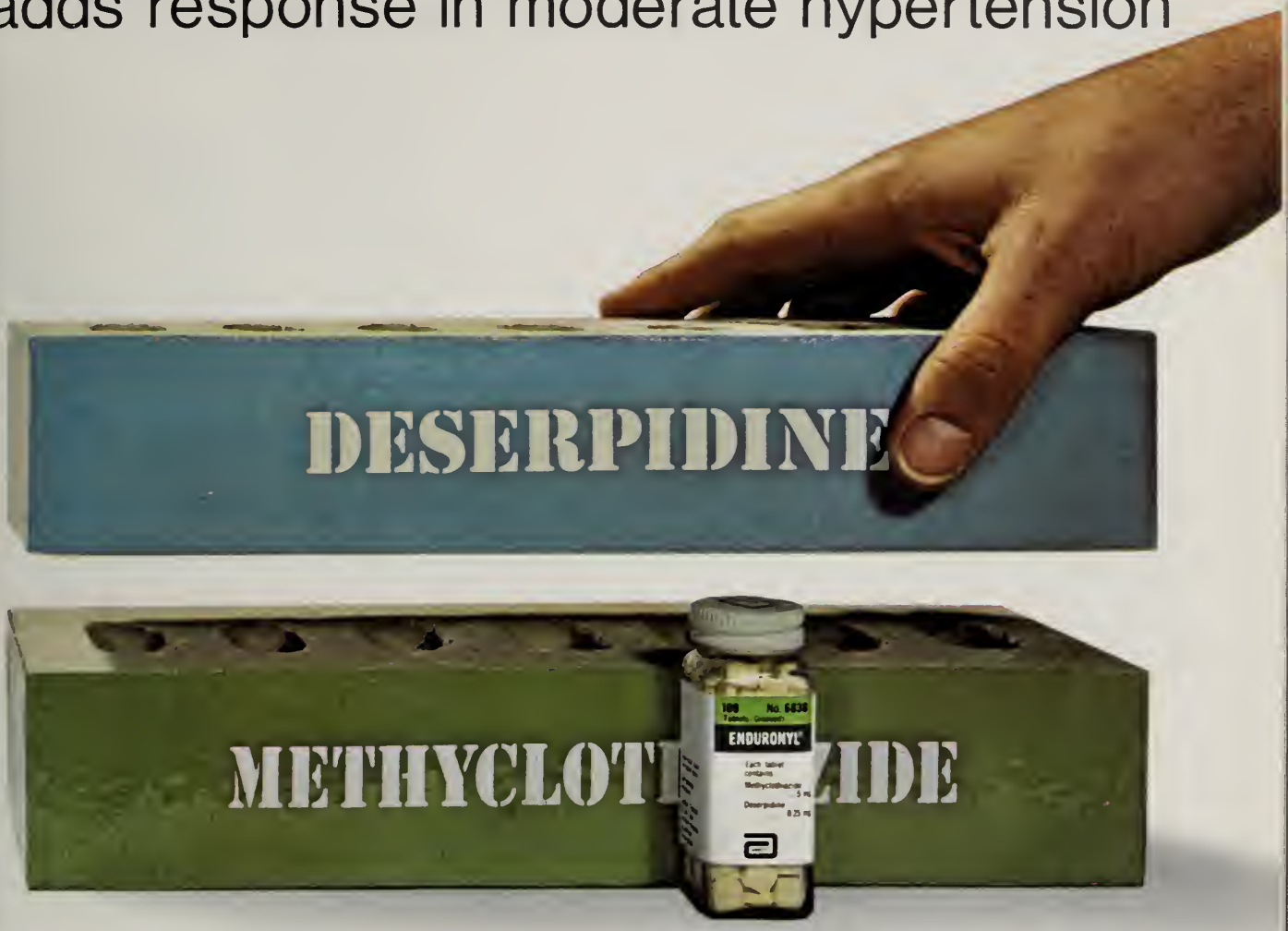
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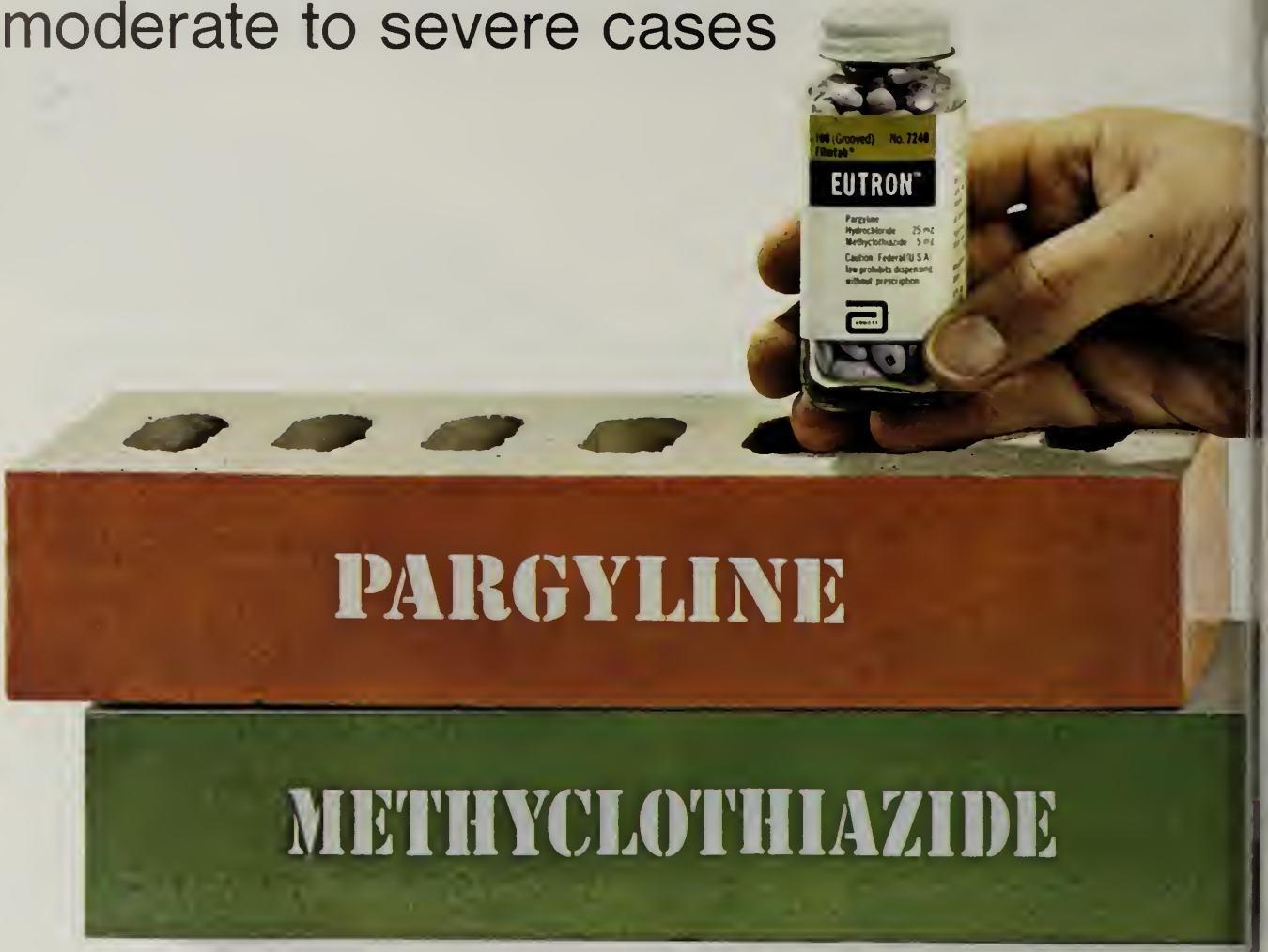
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MILD TO MODERATE TO SEVERE



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Each tablet contains Methyclothiazide 5 mg. with Deserpidine 0.25 mg. or 0.5 mg.

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Contraindications: Sensitivity to thiazides; severe renal disease (except nephrosis) or shutdown; severe hepatic disease or impending hepatic coma (hepatic coma due to hypokalemia has been reported in patients on thiazides). Do not use Enduronyl in severe mental depression, suicidal tendencies, active peptic ulcer, or ulcerative colitis.

Warnings: Consider possible sensitivity where there is history of allergy or asthma. If added potassium is indicated, dietary supplementation is recommended. Reserve enteric-coated potassium tablets for cautious use only when necessary, as they may induce serious or fatal small bowel lesions (stenosis with or without ulceration), cause obstruction, hemorrhage, and perforation often requiring surgery; discontinue them immediately if abdominal pain, distention, nausea, vomiting, or g.i. bleeding occurs. Neither Enduron nor Enduronyl contains added potassium.

Precautions: Use thiazides cautiously in severe renal dysfunction, impaired hepatic function or progressive liver disease; also in pregnancy (bone marrow depression, thrombocytopenia, and altered carbohydrate metabolism have been reported in certain newborn). In surgery, thiazides may reduce response to vasopressors, and increase response to tubocurarine. Antihypertensive response may be enhanced following sympathectomy. Watch for electrolyte imbalance (e.g., hyponatremia) in all patients. In hypokalemia (especially in digitalized patients) give supplemental potassium. In hypochloremic alkalosis, give supplemental chloride.

Use rauwolfias with caution in patients with history of peptic ulcer. Rauwolfias with anesthetics may produce hypotension and bradycardia. Discontinue Enduronyl two weeks before elective surgery. Consider vagal blocking agents during emergency surgery. In epilepsy, adjust anticonvulsant dosage. In electroshock, shorten stimulus strength and duration. In occasional patients with depressive tendencies, rauwolfias may precipitate severe mental depression that usually disappears when drug is stopped.

Adverse Reactions: Thiazide reaction include blood dyscrasias (thrombocytopenia with purpura, agranulocytosis, aplastic anemia); elevation of BUN, serum uric acid or blood sugar; anorexia, nausea, vomiting, diarrhea, headache, dizziness, paresthesia, weakness, skin rash, photosensitivity, jaundice, symtomatic gout, and pancreatitis. Cutaneous vasculitis in the elderly has been reported with other thiazides. Adverse effects with deserpidine are qualitatively similar to those with reserpine, but their incidence is lower. These include nasal stuffiness, abdominal cramps or diarrhea, nausea, headache, weight gain, reduced libido and potency, peptic ulcer aggravation, epistaxis, skin eruption, asthma in susceptible patients, electrolyte imbalance, excessive salivation, and a reversible Parkinson's syndrome. Excessive drowsiness, fatigue, weakness, and nightmares may signal mental depression. Thrombocytopenia, purpura, and a symptom manifested by dull sensorium, deafness, uveitis, glaucoma, and optic atrophy are rare allergic reactions to other rauwolfias. Hypotension from antihypertensive agents may precipitate angina attacks in susceptible individuals. Usually adverse reactions disappear when drug is withdrawn.

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Each tablet contains Pargyline Hydrochloride 25 mg. with Methyclothiazide 5 mg.

Indications—Moderate to severe hypertension.

Contraindications—Pheochromocytoma, paranoid schizophrenia, hyperthyroidism and advanced renal failure. Not recommended in malignant hypertension, children under 12, pregnant patients.

Do not use with: centrally or peripherally acting sympathomimetic drugs; foods high in tyramine (e.g., aged and natural cheeses); parenteral reserpine or guanethidine; imipramine, amitriptyline, desipramine, nortriptyline or their analogues; other monoamine oxidase inhibitors; methyldopa or dopamine; separate Eutron and these agents by two weeks.

Sensitivity to thiazides; severe renal disease (except nephrosis) or shutdown; severe hepatic disease; impending hepatic coma from thiazide-induced hypokalemia.

Warnings—Patients: 1. No other drugs (particularly "cold preparations" and antihistamines), cheese or alcohol without physician's consent. 2. Promptly report orthostatic symptoms, severe headache, other unusual symptoms. 3. Angina pectoris or coronary artery disease patients must not increase physical activity with improved anginal symptoms or well-being.

Physicians: 1. Use antihistamines, hypnotics, sedatives, tranquilizers and narcotics (meperidine contraindicated) cautiously in reduced doses. 2. Stop Eutron two or more weeks before elective surgery; in emergency surgery reduce premedication (narcotics, sedatives, analgesics, etc.) to 1/4 to 1/5; carefully adjust anesthetic dosage to patient response. 3. Use cautiously in advanced renal failure. 4. Pargyline may induce hypoglycemia. 5. Consider possible sensitivity reactions when a history of allergy or asthma is present. 6. If potassium is indicated, dietary supplement is recommended; enteric-coated potassium tablets may induce serious or fatal small bowel lesions (stenosis with or without ulceration), cause obstruction, hemorrhage, and perforation frequently requiring surgery; discontinue medication immediately if abdominal pain, distention, nausea, vomiting or gastrointestinal bleeding occurs; Eutron does not contain added potassium. 7. Possible systemic lupus erythematosus has been reported for thiazides.

Precautions—Pargyline: Use cautiously at reduced dosage: caffeine, alcohol, antihistamines, barbiturates, chloral hydrate, other hypnotics, sedatives, tranquilizers, narcotics. Periodically do urinalyses, blood counts, liver function tests, etc. Use with caution in liver disease. Watch for orthostatic hypotension, especially in impaired circulation (e.g., angina pectoris, coronary artery disease, cerebral arteriosclerosis); also, augmented hypotension in concomitant febrile illnesses. Reduce or discontinue if hypotension is severe. In impaired renal function watch for cumulative drug effects, elevated BUN and other evidence of progressive renal failure; withdraw drug if these persist. In surgery increased central depressant response (hypotension and increased sedative effect) can be controlled by (1) discontinuing at least two weeks prior; (2) in emergency surgery lowering dose of premedication; (3) when necessary, administering a vasopressor. Do not use in hyperactive and hyperexcitable patients. Pargyline may unmask severe psychotic symptoms where emotional problems pre-exist. Use cautiously in Parkinsonism, especially with antiparkinsonian agents. In prolonged therapy, examine for change in color perception, visual fields, fundi and visual acuity. Also, prolonged therapy has made certain patients refractory to nerve blocking effects of local anesthetics.

Methyclothiazide: Use cautiously in severe renal dysfunction, impaired hepatic function or progressive liver disease; also in pregnancy (bone marrow depression, thrombocytopenia, and altered carbohydrate metabolism have been reported in certain newborn). In surgery thiazide may reduce vasopressor response and increase tubocurarine response. Antihypertensive response may be enhanced following sympathectomy. Watch for electrolyte imbalance (e.g., hyponatremia). Give supplemental chloride if hypochloremic alkalosis occurs and supplemental potassium if hypokalemia occurs (especially in digitalized patients). Thiazides may decrease serum P.B.I. without signs of thyroid disturbance.

Adverse Reactions — Pargyline: Orthostatic hypotension and associated symptoms, mild constipation, fluid retention, edema, dry mouth, sweating, increased appetite, arthralgia, nausea, vomiting, headache, insomnia, difficult in micturition, nightmares, impotence, delayed ejaculation, rash, purpura, weight gain, hyperexcitability, increased neuromuscular activity and other extrapyramidal symptoms. Drug fever is extremely rare. Reduction in blood sugar and hypoglycemic effects are possible. Congestive heart failure has been reported in a few patients with reduced cardiac reserve.

Methyclothiazide: Blood dyscrasias (thrombocytopenia with purpura, agranulocytosis, aplastic anemia); elevation of BUN, blood sugar or serum uric acid (gout may be induced); anorexia, nausea, vomiting, diarrhea, headache, dizziness, paresthesia, weakness, skin rash, photosensitivity, jaundice and pancreatitis. Cutaneous vasculitis in elderly patients has been reported with other thiazides.

If side effects are severe or persist, reduce dosage or withdraw drug. 804438R

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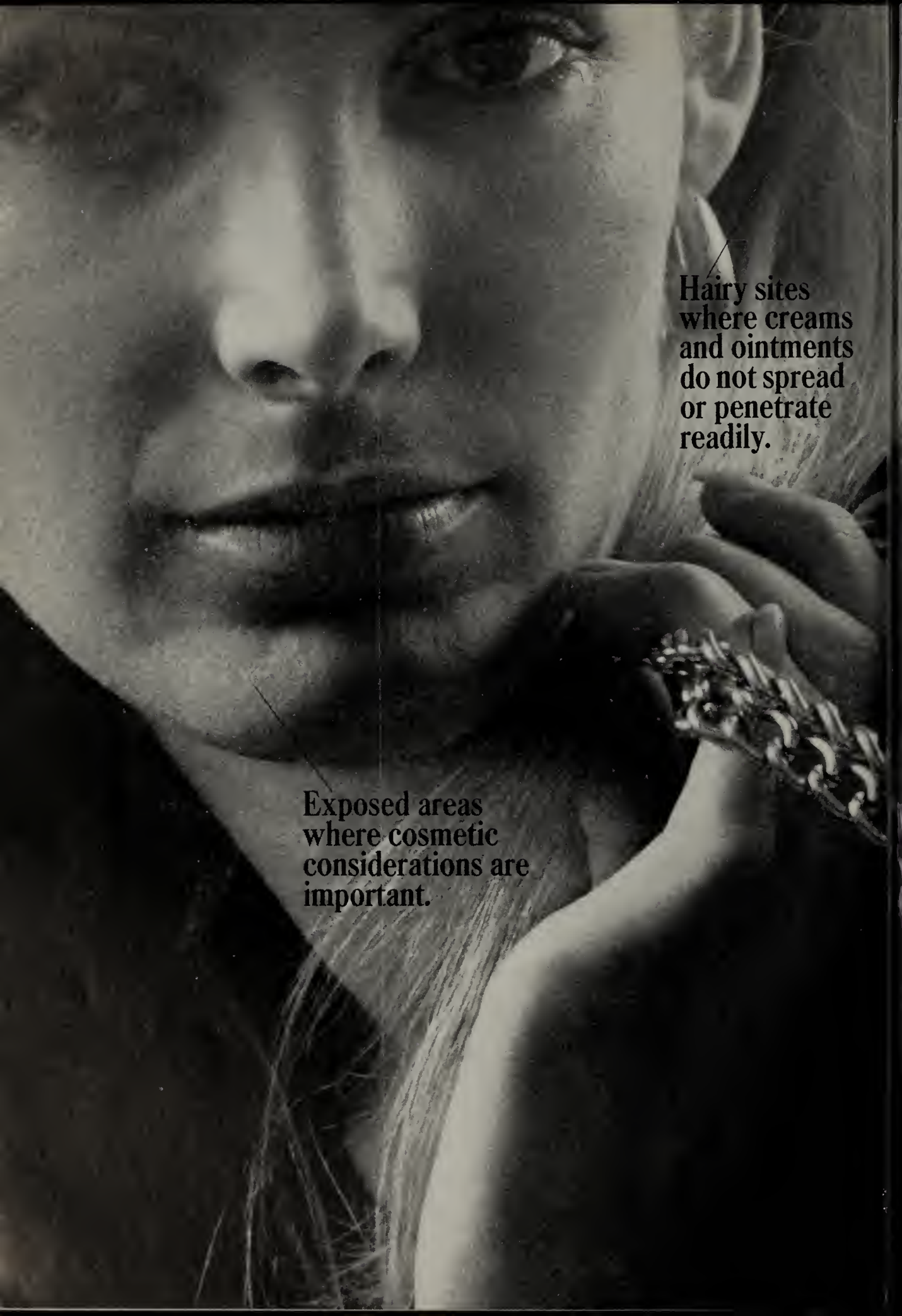
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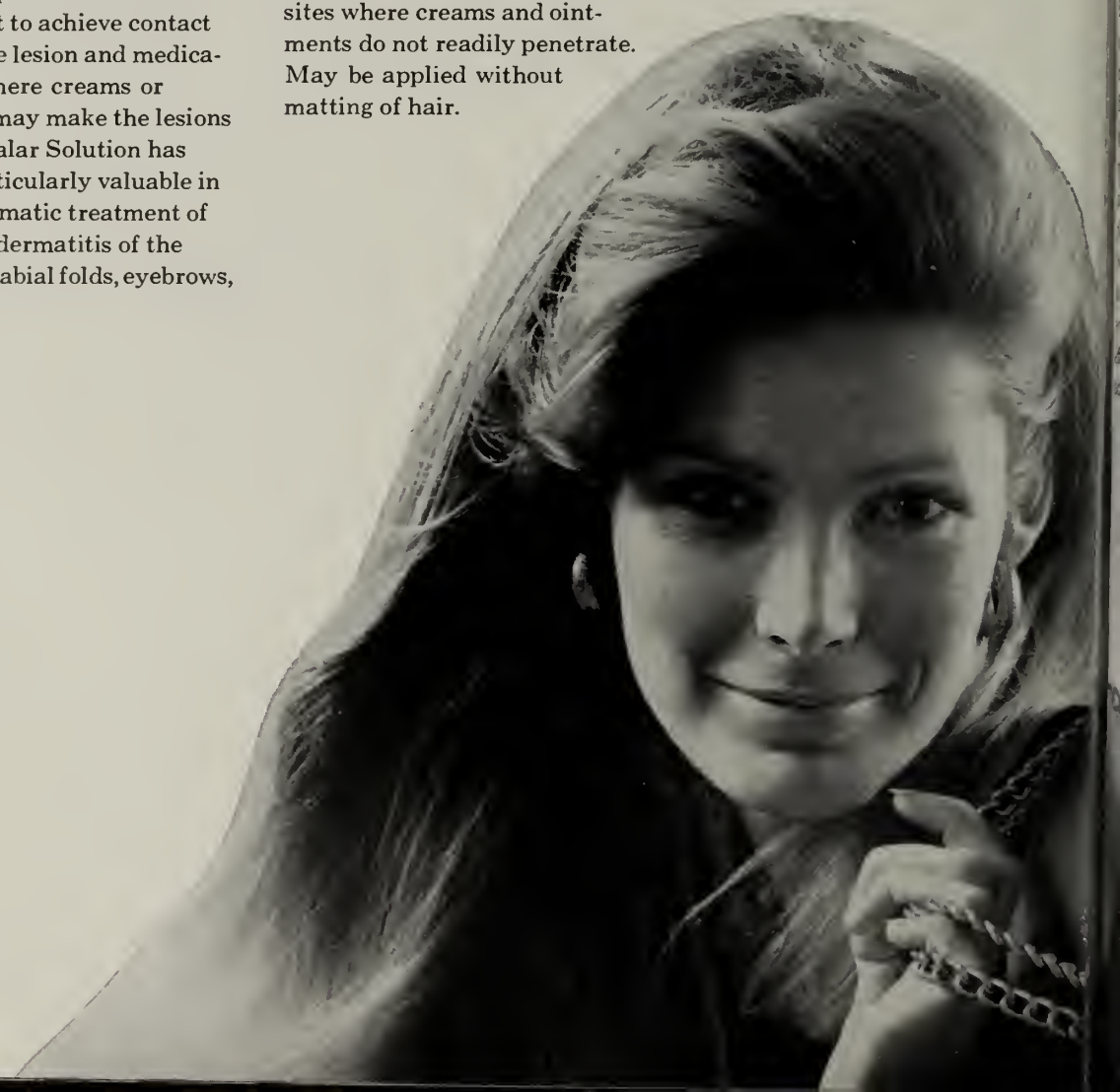
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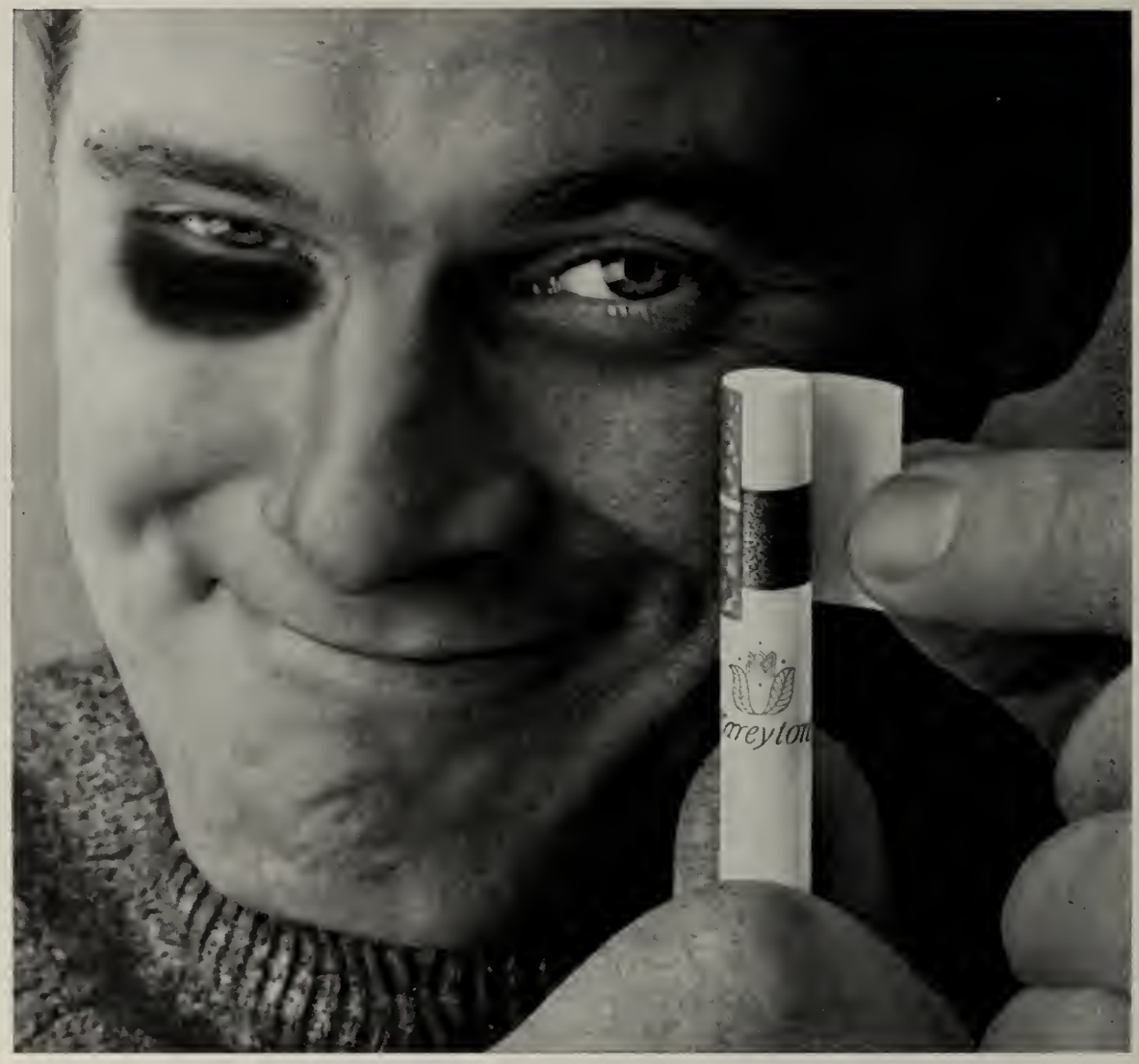
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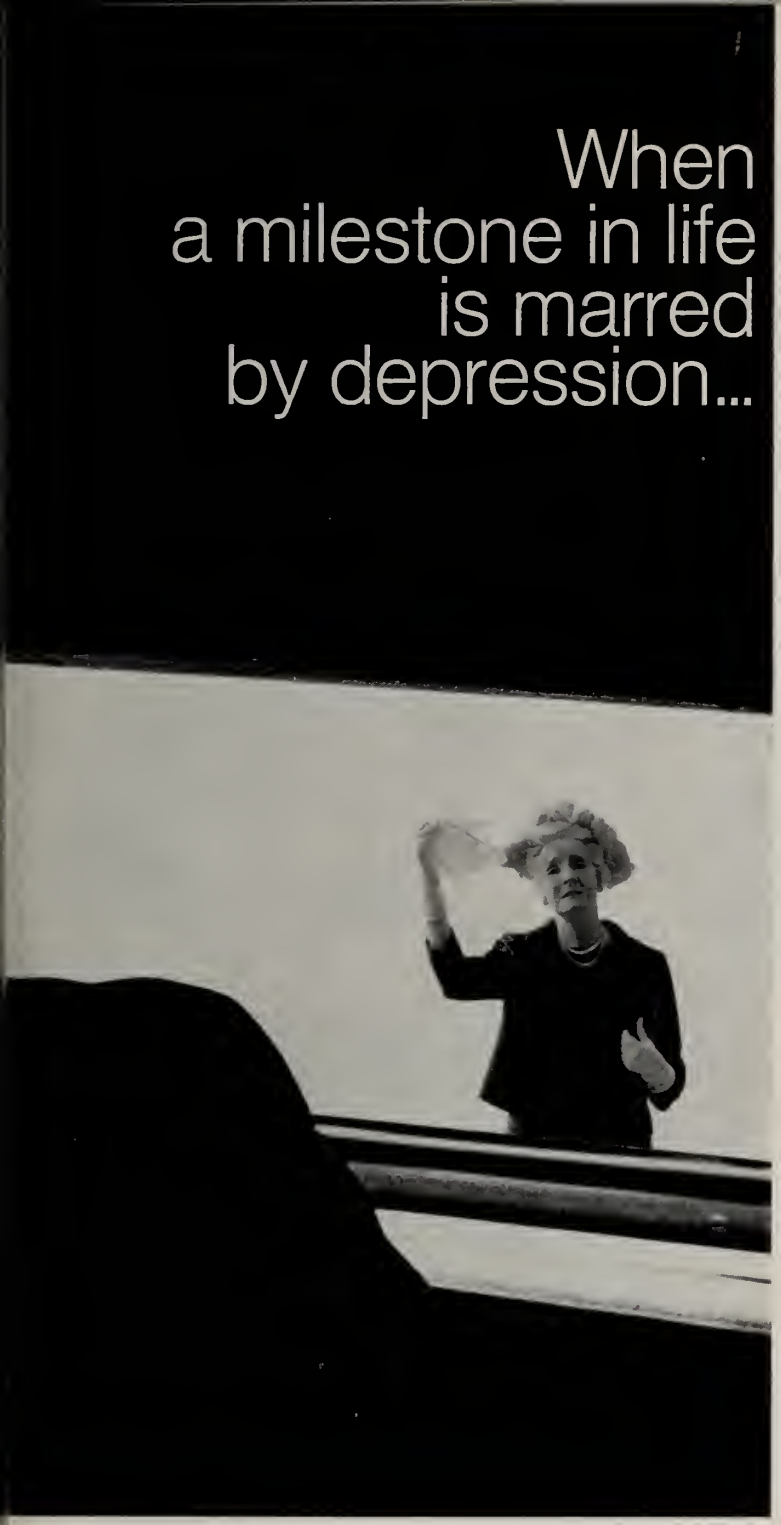
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Indications: Tofrānil is recommended for the treatment of depressive states of diverse psychopathology.
Contraindications: The concomitant use of Tofrānil and monoamine oxidase inhibiting (M.A.O.I.) compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur. Potentiation of adverse effects can be serious or even fatal. An interval of at least 7 days after M.A.O.I. therapy has been discontinued should be allowed before Tofrānil may be substituted. Initial Tofrānil dosage should be low, increases should be gradual, and the patient's progress should be carefully observed.
Warning: Clinical reports have suggested that there may be a risk of teratogenesis associated with the use of this compound during the first trimester of pregnancy. Unless, in the opinion of the prescribing physician, the potential benefits outweigh the possible risks, Tofrānil should not be used during the first trimester of pregnancy.
Cardiovascular complications, including myocardial infarction and arrhythmias, have occasionally occurred in susceptible individuals. Patients with cardiovascular disease should be given the drug only under careful observation and in low dosage.
Precautions: Since suicide is always a possibility in severely depressed patients and one which may persist until significant remission occurs, such patients should be carefully supervised during early treatment with Tofrānil. Some severely depressed patients may also require hospitalization and/or concomitant electroconvulsive therapy.
Because of its anticholinergic effect, caution should be observed in prescribing Tofrānil for patients with increased intraocular pressure.
In rare instances, transient cardiac arrhythmias have occurred in hyperthyroid patients and in patients receiving thyroid medication when Tofrānil was added to the regimen.
Imipramine may block the pharmacologic activity of guanethidine and other related adrenergic neuron-blocking agents.
The drug is not recommended at the present time in patients under 12 years of age.
Adverse Reactions: Dryness of the mouth, tachycardia, constipation, disturbances of accommodation, sweating, dizziness, weight gain, urinary frequency or retention, nausea and vomiting, peripheral neuritis, mild parkinson-like syndrome, tremors, rare cases of falling in elderly patients, confusional states (with such symptoms as hallucinations and disorientation), activation of psychosis in schizophrenics and agitation (includ-

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In patients with cardiovascular disease, hyperthyroidism or increased intraocular pressure; or in those receiving anticholinergics (including antiparkinsonism agents), thyroid medication, or antihypertensive adrenergic neuron-blocking agents; and in those in their first trimester of pregnancy, the special precautions listed in the prescribing information should be carefully observed.

Toxic reactions severe enough to require discontinuation of Tofrānil are uncommon. However, for complete details, please refer to the full prescribing information.

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ing hypomanic and manic episodes) which may require dosage reduction and/or addition of a tranquilizer or temporary discontinuation of the drug, epileptiform seizures, orthostatic hypotension and substantial blood pressure fall in hypertensive patients, purpura, transient jaundice, bone marrow depression including agranulocytosis, sensitization and skin rash including photosensitization, eosinophilia, and mild withdrawal symptoms on sudden discontinuation after prolonged treatment with high doses. Occasional hormonal effects (impotence, decreased libido, and estrogenic effects) may be observed. Atropine-like effects may be more pronounced (e.g. paralytic ileus) in susceptible patients and in those using anticholinergic agents (including antiparkinsonism drugs).
Outpatient Adult Dosage: Initially, 75 mg. daily, increased, if necessary, to 150 or 200 mg. Maintenance dosage may be lower, 50 to 150 mg. daily, if possible.
Geriatric and Adolescent Dosage: Initially, 30 or 40 mg. daily, which may be increased according to response and tolerance. It is usually unnecessary to exceed 100 mg. daily.
A lag in therapeutic response, lasting from a few days to a few weeks, should be expected. When dosage recommendations are already being followed, increasing the dosage does not normally shorten this latency period and may increase the incidence of adverse reactions.
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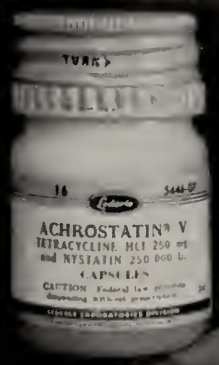
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Precautions: Some individuals may experience drowsiness, anorexia, and slight gastric distress. If excessive drowsiness occurs, it may be necessary to increase the interval between doses. Persons on full dosage should not operate any vehicle. Use may result in overgrowth of nonsusceptible organisms. If infections appear during therapy, appropriate measures should be taken. Infections caused by beta-hemolytic streptococci should be treated for at least 10 full days to help prevent rheumatic fever or acute glomerulonephritis. Use of tetracycline during tooth development may cause discoloration of teeth.
Adverse Reactions: Gastrointestinal—anorexia, nausea, vomiting, diarrhea, stomatitis, glossitis, enterocolitis, pruritus ani. Skin—maculopapular and erythematous rashes (a case of exfoliative dermatitis has been reported); photosensitivity; onycholysis and discoloration of nails (rare). Kidney—rise in BUN, apparently dose related. Hypersensitivity reactions—urticaria, angioneurotic edema, anaphylaxis. In young infants, bulging fontanels following full therapeutic dosage has been reported. This has disappeared rapidly when drug was discontinued. Teeth—dental staining (yellow-brown) in children of mothers given tetracycline during the latter half of pregnancy and in children given the drug during the neonatal period, infancy, and early childhood. Enamel hypoplasia has been seen in a few children. Blood—anemia, thrombocytopenic purpura, neutropenia, eosinophilia. Liver—cholestasis (rare), usually at high dosage. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy.

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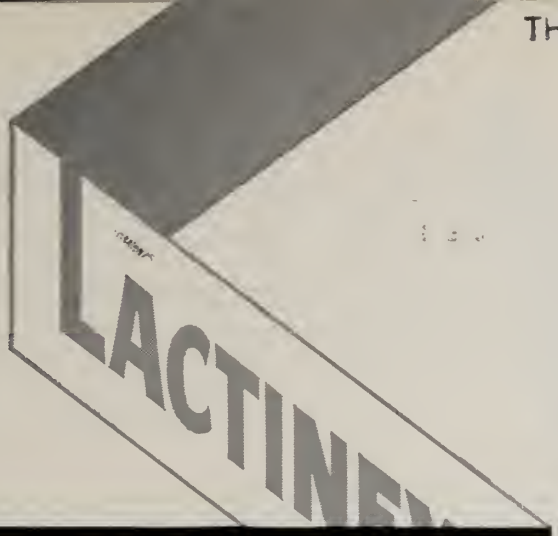
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(1) Siver, R. H.: CMD, *21*:109, September 1954. (2) Frykman, H. H.: Minn. Med., *38*:19-27, January 1955. (3) McGivney, J.: Tex. State Jour. Med., *51*:16-18, January 1955. (4) Quehl, T. M.: Jour. of Florida Acad. Gen. Prac., *15*:15-16, October 1965. (5) Weekes, D. J.: N.Y. State Jour. Med., *58*:2672-2673, August 1958. (6) Weekes, D. J.: EENT Digest, *25*:47-59, December 1963. (7) Abbott, P. L.: Jour. Oral Surg., Anes., & Hosp. Dental Serv., 310-312, July 1961. (8) Rapoport, L. and Levine, W. I.: Oral Surg., Oral Med. & Oral Path., *20*:591-593, November 1965.

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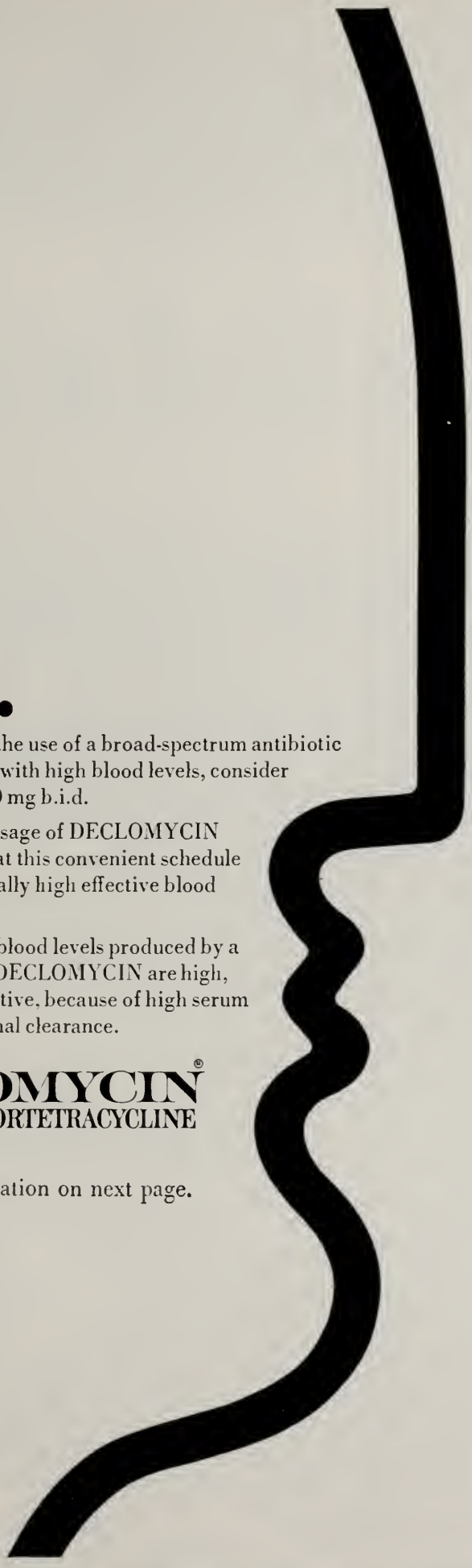
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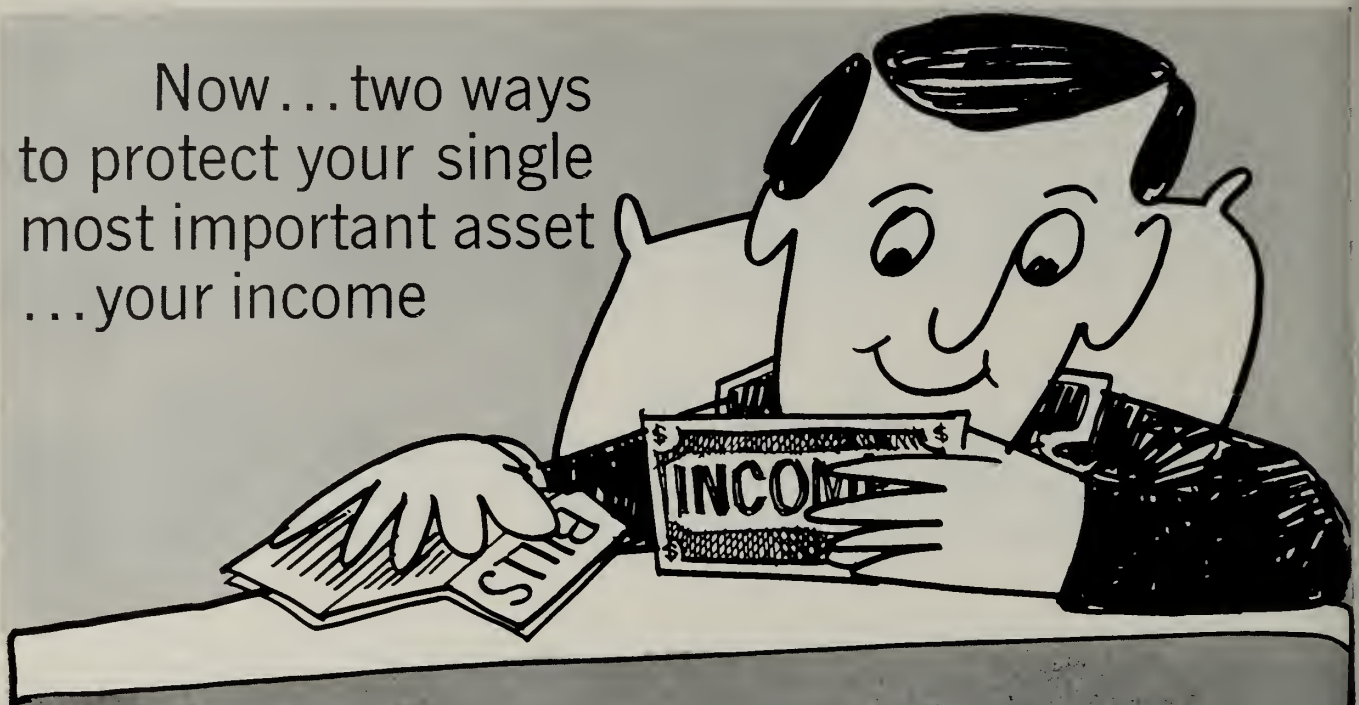
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Contraindications: Edema; danger of cardiac decompensation; history or symptoms of peptic ulcer; renal, hepatic or cardiac damage; history of drug allergy; history of blood dyscrasia. The drug should not be given when the patient is senile or when other potent drugs are given concurrently. Large doses of Butazolidin alka are contraindicated in glaucoma.

Warning: If coumarin-type anticoagulants are given simultaneously, watch for excessive increase in prothrombin time. Instances of severe bleeding have occurred. Persistent or severe dyspepsia may indicate peptic ulcer; perform upper gastrointestinal x-ray diagnostic tests if drug is continued. Pyrazole compounds may potentiate the pharmacologic action of sulfonylurea, sulfonamide-type agents and insulin. Carefully observe patients receiving such therapy. Use with caution in the first trimester of pregnancy and in patients with thyroid disease.

Precautions: Before prescribing, carefully select patients, avoiding those responsive to routine measures as well as contraindicated patients. Obtain a detailed history and a complete physical and laboratory examination, including a blood count. The patient should not exceed recommended dosage, should be closely supervised and should be warned to discontinue the drug and report immediately if fever, sore throat, or mouth lesions (symptoms of blood dyscrasia); sudden weight gain (water retention); skin reactions; black or tarry stools or other evidence of intestinal hemorrhage occur. Make complete blood counts at weekly intervals during early therapy and at 2-week intervals thereafter. Discontinue the drug immediately and institute countermeasures if the white count changes significantly, granulocytes decrease, or immature forms appear. Use greater care in the elderly and in hypertensives.

Adverse Reactions: The more common are nausea and edema. Swelling of the ankles or face may be minimized by withholding dietary salt, reduction in dosage or use of diuretics. In elderly patients and in those with hypertension the drug should be discontinued with the appearance of edema. The drug has been associated with peptic ulcer and may reactivate a latent peptic ulcer. The patient should be instructed to take doses immediately before or after meals or with milk to minimize gastric upset. Drug rash occasionally occurs. If it does, promptly discontinue the drug. Agranulocytosis, exfoliative dermatitis, Stevens-Johnson syndrome, Lyell's syndrome (toxic necrotizing epidermolysis), or a generalized allergic reaction similar to serum sickness may occur and require permanent withdrawal of medication. Agranulocytosis can occur suddenly in spite of regular, repeated normal white counts. Stomatitis, salivary gland enlargement, vomiting, vertigo and languor may occur. Leukemia and leukemoid reactions have been reported. While not definitely attributable to the drug, a causal relationship cannot be excluded. Thrombocytopenic purpura and aplastic anemia may occur. Confusional states, agitation, headache, blurred vision, optic neuritis and transient hearing loss have been reported, as have hyperglycemia, hepatitis, jaundice, hypersensitivity angiitis, pericarditis and several cases of anuria and hematuria. With long-term use, reversible thyroid hyperplasia may occur infrequently. Moderate lowering of the red cell count due to hemodilution may occur.



Dosage in Rheumatoid Arthritis: Initial: 3 to 6 capsules daily in 3 or 4 equal doses. Trial period: 1 week. Maintenance dosage should not exceed 4 capsules daily; response is often achieved with 1 or 2 capsules daily.

In selecting appropriate dosage in any specific case, consideration should be given to the patient's weight, general health, age and any other factors influencing drug response. (B)46-070-A

For complete details, please see full Prescribing Information.

Butazolidin® alka Geigy

Capsules
phenylbutazone, 100 mg.; dried aluminum hydroxide gel, 100 mg.; magnesium trisilicate, 150 mg.; homatropine methylbromide, 1.25 mg.

Geigy Pharmaceuticals
Division of Geigy Chemical Corporation
Ardsley, New York 10502

BU 5463 R

When it's more than a bad cold



your patient can feel better while he's getting better

Achrocidin®

Tetracycline HCl—Antihistamine—Analgesic Compound

Each tablet contains: ACHROMYCIN® Tetracycline HCl 125 mg.; Phenacetin 120 mg.; Caffeine 30 mg.; Salicylamide 150 mg.; Chlorothen citrate 25 mg.

In bacterial/allergic u.r.i., ACHROCIDIN brings the treatment together in a single prescription—prompt relief of headache and congestion together with effective control of the tetracycline-sensitive organisms frequently responsible for complications leading to prolonged disability in the susceptible patient.

For children and elderly patients you may prefer caffeine-free ACHROCIDIN Syrup. Each 5 cc contains: ACHROMYCIN (Tetracycline) equivalent to Tetracycline HCl 125 mg.; Phenacetin 120 mg.; Salicylamide 150 mg.; Ascorbic Acid (C) 25 mg.; Pyrilamine Maleate 15 mg.

Average adult dosage: 2 tablets four times daily, given at least one hour before, or two hours after meals.
Contraindications: History of hypersensitivity to any component.
Warning: If renal impairment exists, even usual doses may lead to liver toxicity. Under such conditions, lower than usual doses are indicated and, if therapy is prolonged, serum level determinations may be advisable. Hypersensitive individuals may develop a photodynamic reaction to natural or artificial sunlight during use. Individuals with a history of photosensitivity reactions should avoid direct exposure while under treatment, which should be discontinued at first evidence of skin discomfort.
Precautions: Some individuals may experience drowsiness, anorexia, and slight gastric distress. If excessive drowsiness occurs, it may be necessary to increase the interval between doses. Persons on full dosage should not operate any vehicle. Use may result in overgrowth of nonsusceptible organisms. If infections appear during therapy, appropriate measures should be taken. Infections caused by beta-hemolytic streptococci should be treated for at least 10 full days to help prevent rheumatic fever or acute glomerulonephritis. Use of tetracycline during tooth development may cause discoloration of teeth.
Adverse Reactions: Gastrointestinal—anorexia, nausea, vomiting, diarrhea, stomatitis, glossitis, enterocolitis, pruritus ani. Skin—maculopapular and erythematous rashes (a case of exfoliative dermatitis has been reported); photosensitivity; onycholysis and discoloration of nails (rare). Kidney—rise in BUN, apparently dose related. Hypersensitivity reactions—urticaria, angioneurotic edema, anaphylaxis. In young infants, bulging fontanels following full therapeutic dosage has been reported. This has disappeared rapidly when drug was discontinued. Teeth—dental staining (yellow-brown) in children of mothers given tetracycline during the latter half of pregnancy and in children given the drug during the neonatal period, infancy, and early childhood. Enamel hypoplasia has been seen in a few children. Blood—anemia, thrombocytopenic purpura, neutropenia, eosinophilia. Liver—cholestasis (rare), usually at high dosage. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy.

Lederle

350-8

The AMBAR SCRAPBOOK of

Obesity Oddities

FACT & LEGEND



CONTROL FOOD AND MOOD ALL DAY LONG WITH A SINGLE MORNING DOSE

AMBAR #2 EXTENTABS®

methamphetamine HCl 15 mg., phenobarbital 64.8 mg. (1 gr.) (Warning: may be habit forming).

One Ambar Extentab before breakfast can help control most patients' appetite for up to 12 hours. Methamphetamine, the appetite suppressant, gently elevates mood and helps overcome dieting frustrations. Phenobarbital, the sedative in Ambar, controls irritability and anxiety...helps maintain a state of mental calm and equanimity. Both work together to ease the tensions that erode the willpower during periods of dieting.

Also available: Ambar #1 Extentabs®—methamphetamine hydrochloride 10 mg., phenobarbital 64.8 mg. (1 gr.) (Warning: may be habit forming).

BRIEF SUMMARY/**Indications:** Ambar suppresses appetite and helps offset emotional reactions to dieting. **Contraindications:** Hypersensitivity to barbiturates or sympathomimetics; patients with advanced renal or hepatic disease. **Precautions:** Administer with caution in the presence of cardiovascular disease or hypertension. **Side Effects:** Nervousness or excitement occasionally noted, but usually infrequent at recommended dosages. Slight drowsiness has been reported rarely. See package insert for further details.

A. H. ROBINS COMPANY, RICHMOND, VA. 23220 A·H·ROBINS



After the picnic even Gramps Was a victim of intestinal cramps

Parepectolin for quick relief of acute diarrhea
... soothes colicky pain with paregoric*
... consolidates fluid stools with pectin
... adsorbs irritants with kaolin,
and protects intestinal mucosa

In elderly patients it is particularly important to stop the diarrhea fast. Parepectolin helps you control diarrhea promptly and gain the patient's confidence until etiology has been determined.

Parepectolin®

Each fluid ounce of creamy white suspension contains:
*Paregoric (equivalent) ..... (1.0 dram) 3.7 ml.
Contains opium (¼ grain) 15 mg. per fluid ounce.
warning: may be habit forming
Pectin ..... (2½ grains) 162 mg.
Kaolin (specially purified) .... (85 grains) 5.5 Gm.
(alcohol 0.69%)
Usual Adult Dose: One or two tablespoonfuls three times daily.

ROER
WILLIAM H. RORER, INC.
Fort Washington, Pa.

mudrane®

for

- EMPHYSEMA
- ASTHMA
- CHRONIC BRONCHITIS
- BRONCHIECTASIS

The fast-disintegrating uncoated tablet gives relief in 15 minutes

Each tablet contains:

Potassium Iodide	195 mg.
Aminophylline	130 mg.
Phenobarbital, Caution: May be habit forming...	21 mg.
Ephedrine HCl	16 mg.

FEDERAL LAW PROHIBITS
DISPENSING WITHOUT PRESCRIPTION

Precautions: Usual for aminophylline-ephedrine-phenobarbital. Iodides may cause nausea, long use may cause goiter. Discontinue if symptoms of iodism develop.

Iodide contraindications: tuberculosis, pregnancy.

DOSAGE

One tablet, with full glass of water, 3 or 4 times daily.

Dispensed in bottles of 100 and 1000 tablets.

MUDRANE GG—Formula, dosage and package identical to Mudrane—*except*—100 mg. glyceryl guaiacolate replaces the potassium iodide. The value of Mudrane cannot be enjoyed by a small group in which K.I. is contraindicated. Mudrane GG is prepared for this group.

MUDRANE GG ELIXIR—Four 5 cc teaspoonfuls is equivalent to one Mudrane GG tablet. Dosage adjusted to age and weight of child. Mudrane GG Elixir is for pediatric patients and those who think they cannot swallow tablets. Dispensed in pint and half gallon bottles.

WM. P. POYTHRESS & CO., INC.
RICHMOND, VIRGINIA 23217
Manufacturers of ethical pharmaceuticals since 1856

The muscle relaxant that works before you write a prescription

Relieve painful skeletal muscle spasm in your office in minutes with a single 2 cc. injection of NORFLEX. Then, for sustained relief, write a prescription for NORFLEX tablets, 1 tablet b.i.d.

CONTRAINDICATIONS: Due to its anticholinergic action, NORFLEX should not be used in patients with glaucoma, pyloric or duodenal obstruction, stenosing peptic ulcer, prostatic hypertrophy or obstruction at the bladder neck, cardiospasm (megaesophagus) and myasthenia gravis. Use with caution in patients with tachycardia. Do not use propoxyphene (Darvon®) concurrently.

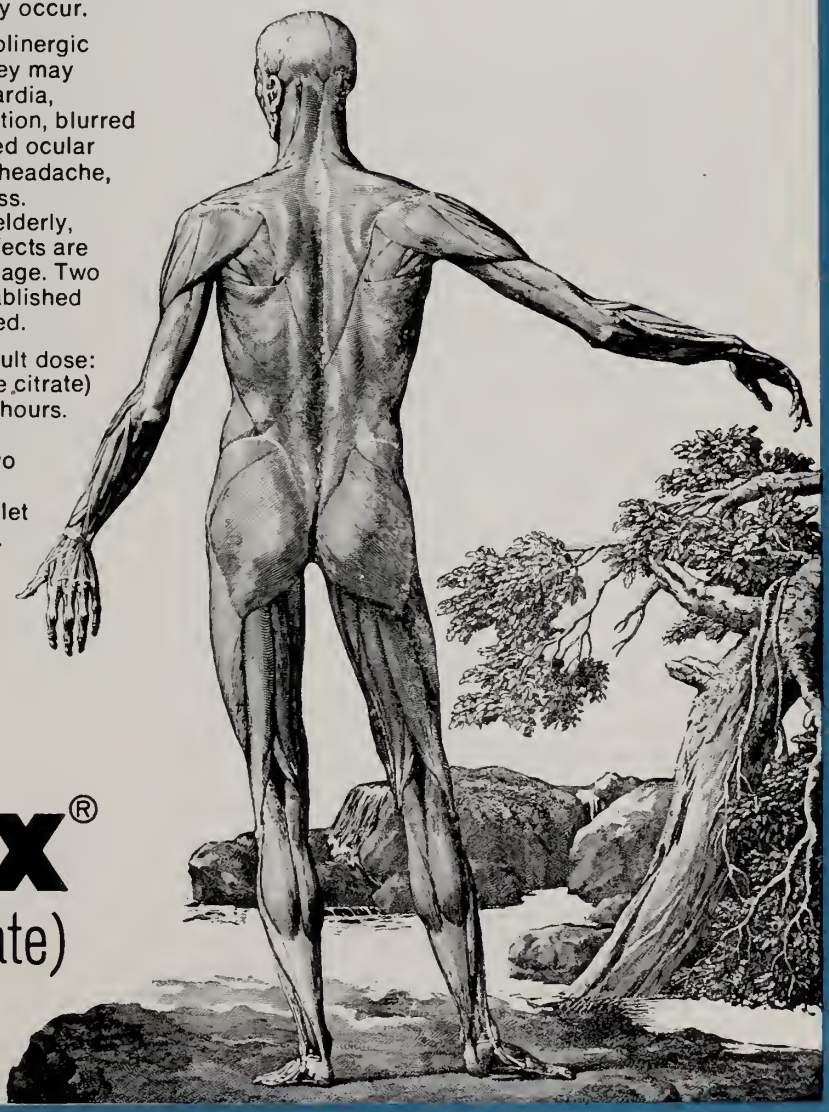
WARNING: Transient lightheadedness or dizziness following NORFLEX-INJECTABLE may occur.

SIDE EFFECTS: Due mainly to anticholinergic action and usually at high dosage. They may include dryness of the mouth, tachycardia, palpitation, urinary hesitancy or retention, blurred vision, dilatation of the pupil, increased ocular tension, weakness, nausea, vomiting, headache, dizziness, constipation, and drowsiness. Infrequently, mental confusion in the elderly, urticaria or other dermatoses. Side effects are usually eliminated by reduction in dosage. Two cases of aplastic anemia, with no established causal relationship, have been reported.

DOSAGE: **INJECTABLE** — Average adult dose: one ampul, 2 cc. (60 mg. orphenadrine citrate) I.M. or I.V. May be repeated every 12 hours. Relief may be maintained with one NORFLEX tablet b.i.d. **TABLETS** — Two tablets per day for adults, one in the morning, one in the evening. Each tablet contains 100 mg. orphenadrine citrate.

For full information, see Package Insert or P.D.R.

Riker Laboratories
Northridge, California 91324



Norflex®
(orphenadrine citrate)

Diarrhea

TROCINATE® 400 MG.

BRAND THIPHENAMIL HCl.

The relief received from the first Trocinate 400 mg. tablet is so prompt that the discomfort of diarrhea ceases to be a bother. May be repeated every four hours.

Upon request, a supply of Trocinate 400 mg. with literature will be sent to physicians for their personal use.

WM. P. POYTHRESS & CO., INC.
RICHMOND, VIRGINIA 23217
Manufacturers of ethical pharmaceuticals since 1856



Whenever the diet is faulty, the appetite poor, or the loss of food is excessive

through vomiting or diarrhea—

Valentine's
MEAT EXTRACT

stimulates the appetite,

increases the flow of digestive juices,

provides: supplementary amounts of vitamins, minerals and soluble proteins,

extra-dietary vitamin B_{12},

protective quantities of potassium, in a palatable and readily assimilated form.

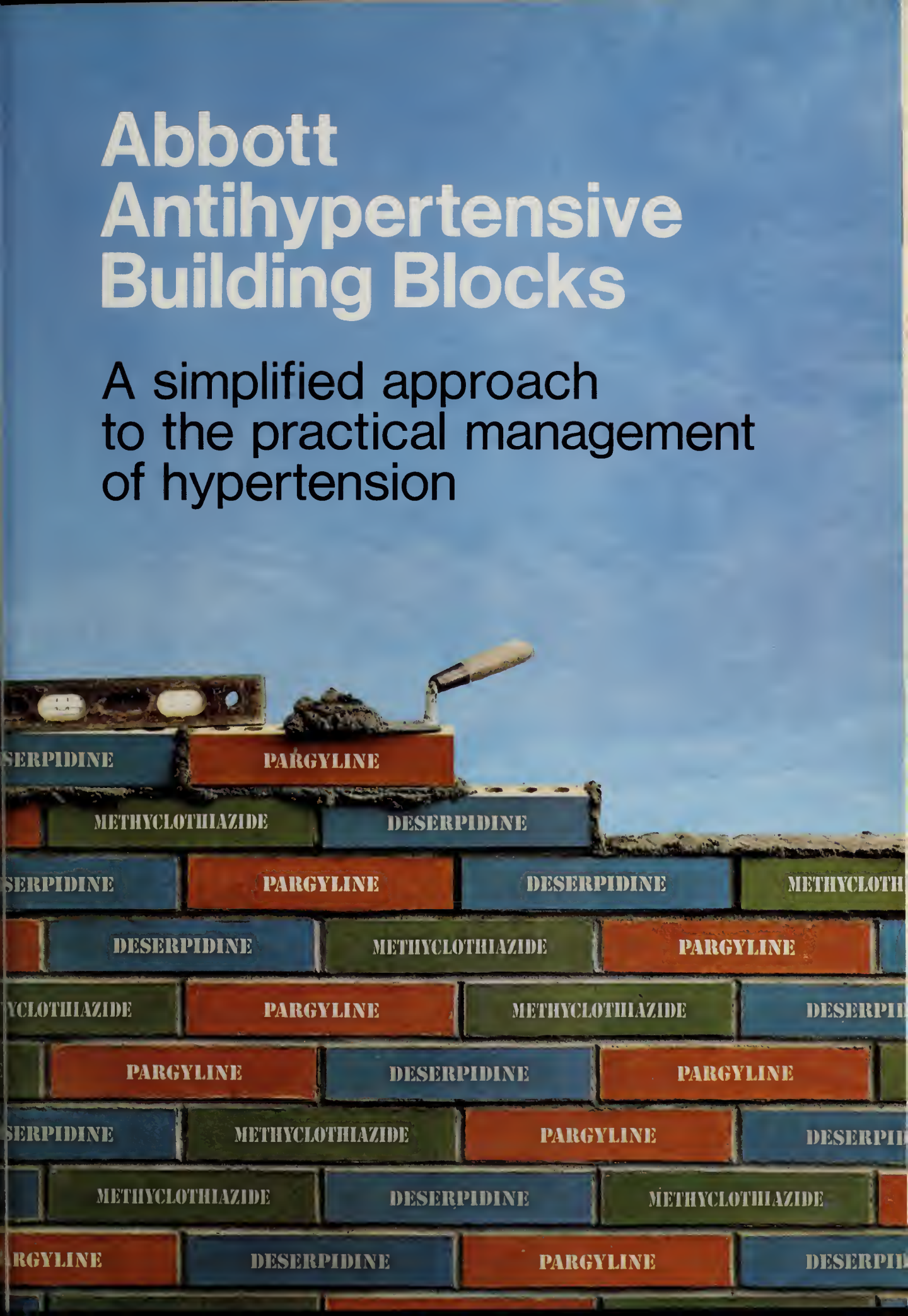
Supplied in bottles of 2 or 6 fluidounces.

DOSAGE *is 1 teaspoonful two or three times daily; two or three times this amount for potassium therapy. Dilute with two or more equal volumes of water.*

VALENTINE Company, Inc.
RICHMOND 21, VIRGINIA

Abbott Antihypertensive Building Blocks

A simplified approach to the practical management of hypertension



Enduron: (methyclothiazide) A basic building block for mild hypertensives



Excellent day-long Na^+ output, yet easy on the K^+

Enduron provides an excellent starting therapy. Your patient's sodium excretion is greatly enhanced. Yet potassium loss is low.

The therapeutic action is smooth, and persists for a full 24 hours. With Enduron you can prescribe convenient once-a-day dosage without skimping your patients on day-long thiazide effectiveness.

Of course, as with all thiazides, supplemental dietary potassium should also be considered.

Use Enduron as a basic therapy in patients with mild to moderate hypertension. A single 5-mg. tablet each day is ample in most cases.

Once a day, every day

ENDURON

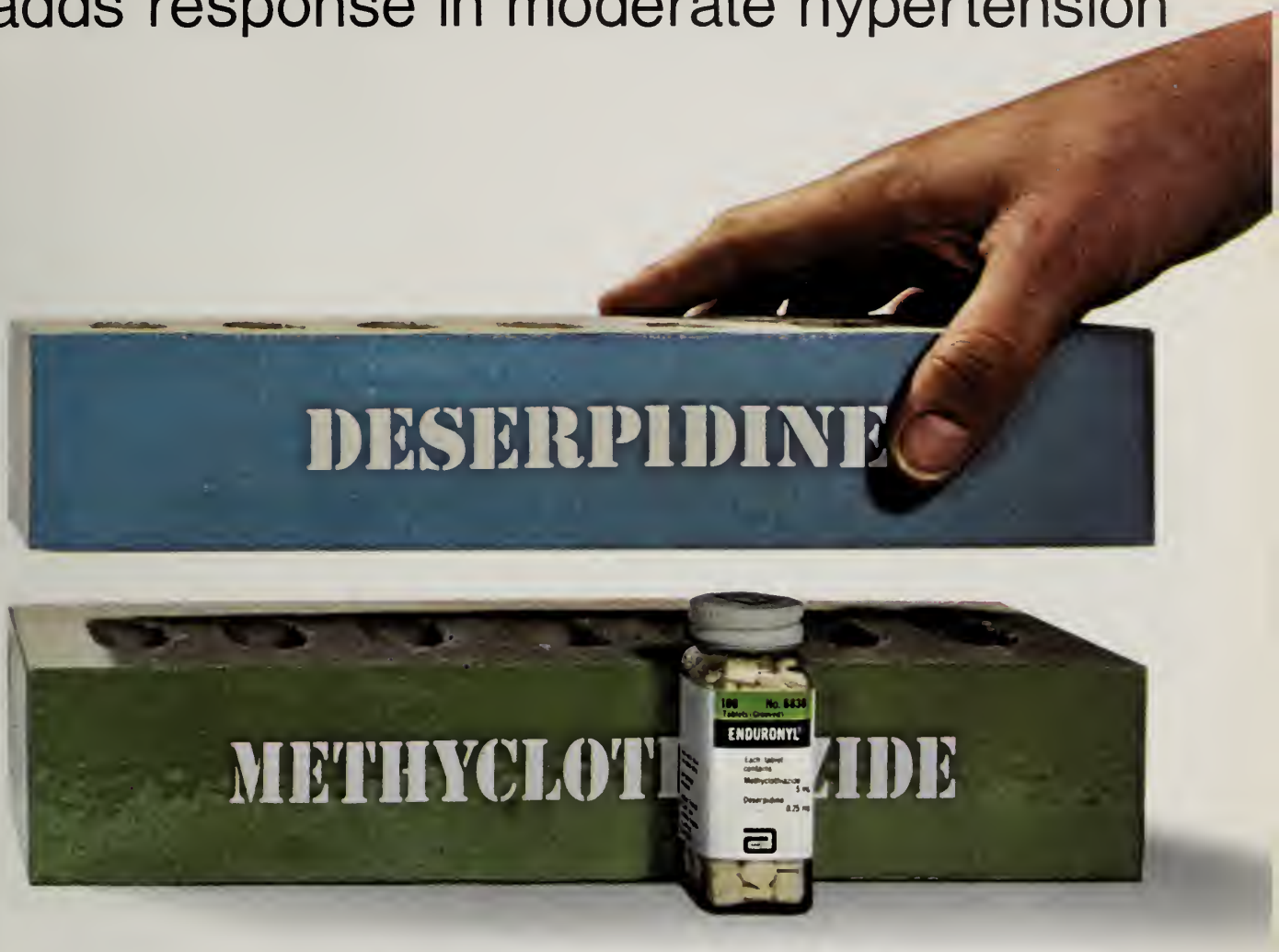
METHYCLOTHIAZIDE

MILD TO MODERATE TO SEVERE



See Brief Summary on final page of advertisement

Enduronyl: Its deserpidine component adds response in moderate hypertension



Less frequent rauwolfia side effects than with reserpine

When you wish to build further response, consider shifting to Enduronyl.

Enduronyl adds a building block of *deserpidine*. This is a purified rauwolfia alkaloid available only from Abbott. It adds good antihypertensive and tranquilizing activity. Yet its incidence of untoward effects, particularly lethargy and depression, is lower than with reserpine.

Enduronyl is available plain or Forte. The latter provides its variation where most helpful, by doubling the deserpidine.

Use Enduronyl for patients throughout the broad range of mild to moderately severe hypertension.

Once a day, every day

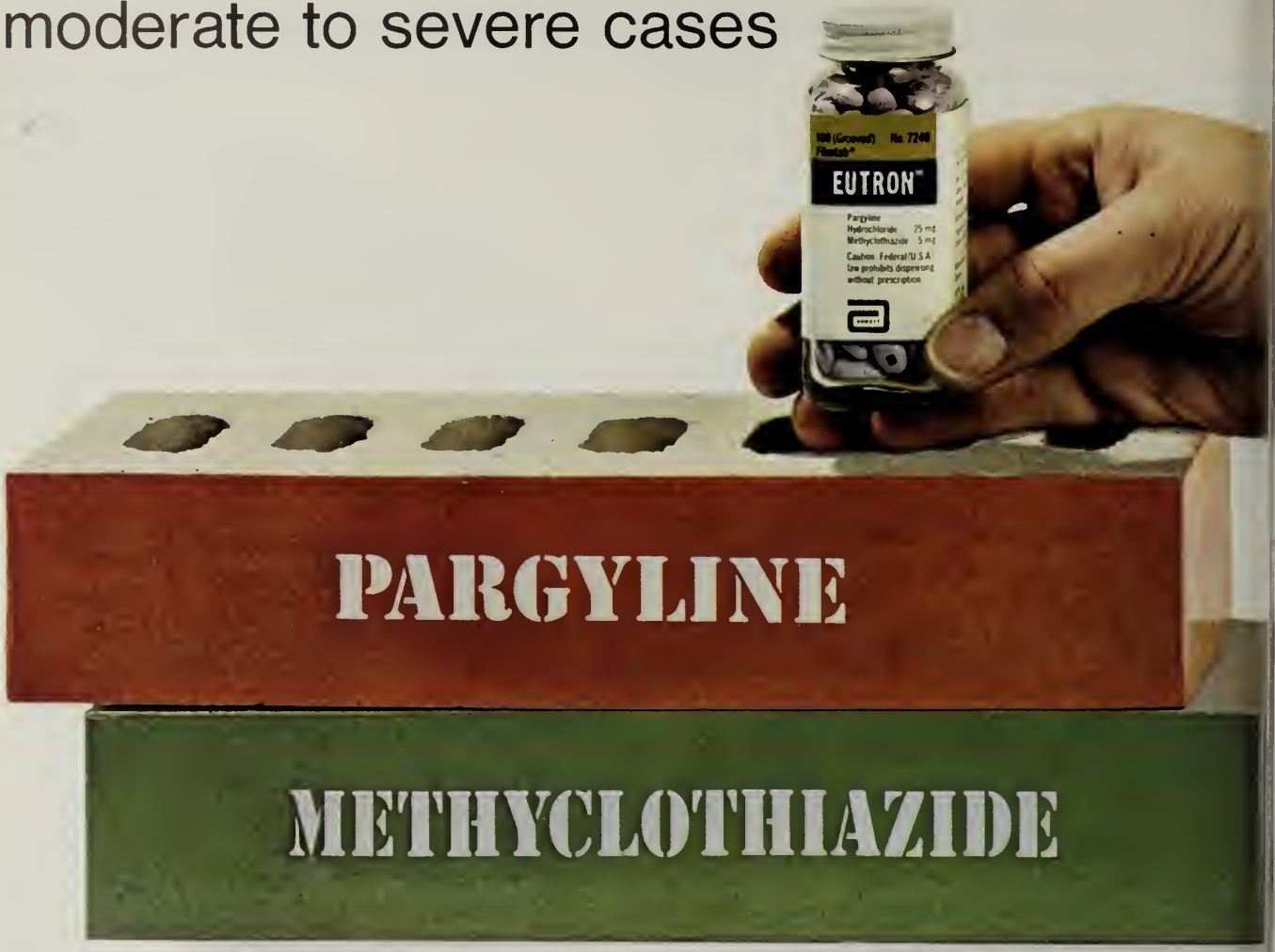
ENDURONYL®

METHYCLOTHIAZIDE 5 mg. with DESERPIDINE 0.25 mg. or (FORTE) 0.5 mg.



See Brief Summary on final page of advertisement

Eutron: A unique combination for handling moderate to severe cases



Affords almost uniform diastolic reduction in all body positions

Eutron lowers diastolic pressures nearly equally, whether your patient is standing up or lying down.

Thus, in clinical trials, average *standing* diastolic readings were reduced from 112 pre-treatment to 90 post-treatment; *sitting* from 115 to 95; and *recumbent* from 112 to 94.

Note that following Eutron, the diastolic reductions were nearly alike in all three body positions.

Use Eutron for managing your moderate to severe cases. Its building blocks enhance each other; hence lesser doses often suffice.

Once a day, every day

EUTRON™

PARGYLINE HYDROCHLORIDE 25 mg.
with METHYCLOTHIAZIDE 5 mg.

MILD TO MODERATE TO SEVERE



See Brief Summary on final page of advertisement

ENDURON® | ENDURONYL®

METHYCLOTHIAZIDE

Each tablet contains
Methyclothiazide 5 mg. with
Deserpidine 0.25 mg. or 0.5 mg.

Indications: Edema and mild to moderate hypertension (Enduron), and mild to moderately severe hypertension (Enduronyl). More potent agents, if added, can be given at reduced dosage.

Contraindications: Sensitivity to thiazides; severe renal disease (except nephrosis) or shutdown; severe hepatic disease or impending hepatic coma (hepatic coma due to hypokalemia has been reported in patients on thiazides). Do not use Enduronyl in severe mental depression, suicidal tendencies, active peptic ulcer, or ulcerative colitis.

Warnings: Consider possible sensitivity where there is history of allergy or asthma. If added potassium is indicated, dietary supplementation is recommended. Reserve enteric-coated potassium tablets for cautious use only when necessary, as they may induce serious or fatal small bowel lesions (stenosis with or without ulceration), cause obstruction, hemorrhage, and perforation often requiring surgery; discontinue them immediately if abdominal pain, distention, nausea, vomiting, or g.i. bleeding occurs. Neither Enduron nor Enduronyl contains added potassium.

Precautions: Use thiazides cautiously in severe renal dysfunction, impaired hepatic function or progressive liver disease; also in pregnancy (bone marrow depression, thrombocytopenia, and altered carbohydrate metabolism have been reported in certain newborn). In surgery, thiazides may reduce response to vasopressors, and increase response to tubocurarine. Antihypertensive response may be enhanced following sympathectomy. Watch for electrolyte imbalance (e.g., hyponatremia) in all patients. In hypokalemia (especially in digitalized patients) give supplemental potassium. In hypochloremic alkalosis, give supplemental chloride.

Use rauwolfias with caution in patients with history of peptic ulcer. Rauwolfias with anesthetics may produce hypotension and bradycardia. Discontinue Enduronyl two weeks before elective surgery. Consider vagal blocking agents during emergency surgery. In epilepsy, adjust anticonvulsant dosage. In electroshock, shorten stimulus strength and duration. In occasional patients with depressive tendencies, rauwolfias may precipitate severe mental depression that usually disappears when drug is stopped.

Adverse Reactions: Thiazide reaction include blood dyscrasias (thrombocytopenia with purpura, agranulocytosis, aplastic anemia); elevation of BUN, serum uric acid or blood sugar; anorexia, nausea, vomiting, diarrhea, headache, dizziness, paresthesia, weakness, skin rash, photosensitivity, jaundice, symtomatic gout, and pancreatitis. Cutaneous vasculitis in the elderly has been reported with other thiazides. Adverse effects with deserpidine are qualitatively similar to those with reserpine, but their incidence is lower. These include nasal stuffiness, abdominal cramps or diarrhea, nausea, headache, weight gain, reduced libido and potency, peptic ulcer aggravation, epistaxis, skin eruption, asthma in susceptible patients, electrolyte imbalance, excessive salivation, and a reversible Parkinson's syndrome. Excessive drowsiness, fatigue, weakness, and nightmares may signal mental depression. Thrombocytopenia, purpura, and a symptom manifested by dull sensorium, deafness, uveitis, glaucoma, and optic atrophy are rare allergic reactions to other rauwolfias. Hypotension from antihypertensive agents may precipitate angina attacks in susceptible individuals. Usually adverse reactions disappear when drug is withdrawn.

EUTRON™

Each tablet contains
Pargyline Hydrochloride 25 mg.
with Methyclothiazide 5 mg.

Indications—Moderate to severe hypertension.

Contraindications—Pheochromocytoma, paranoid schizophrenia, hyperthyroidism and advanced renal failure. Not recommended in malignant hypertension, children under 12, pregnant patients.

Do not use with: centrally or peripherally acting sympathomimetic drugs; foods high in tyramine (e.g., aged and natural cheeses); parenteral reserpine or guanethidine; imipramine, amitriptyline, desipramine, nortriptyline or their analogues; other monoamine oxidase inhibitors; methyldopa or dopamine; separate Eutron and these agents by two weeks.

Sensitivity to thiazides; severe renal disease (except nephrosis) or shutdown; severe hepatic disease; impending hepatic coma from thiazide-induced hypokalemia.

Warnings—Patients: 1. No other drugs (particularly "cold preparations" and antihistamines), cheese or alcohol without physician's consent. 2. Promptly report orthostatic symptoms, severe headache, other unusual symptoms. 3. Angina pectoris or coronary artery disease patients must not increase physical activity with improved anginal symptoms or well-being.

Physicians: 1. Use antihistamines, hypnotics, sedatives, tranquilizers and narcotics (meperidine contraindicated) cautiously in reduced doses. 2. Stop Eutron two or more weeks before elective surgery; in emergency surgery reduce premedication (narcotics, sedatives, analgesics, etc.) to 1/4 to 1/5; carefully adjust anesthetic dosage to patient response. 3. Use cautiously in advanced renal failure. 4. Pargyline may induce hypoglycemia. 5. Consider possible sensitivity reactions when a history of allergy or asthma is present. 6. If potassium is indicated, dietary supplement is recommended; enteric-coated potassium tablets may induce serious or fatal small bowel lesions (stenosis with or without ulceration), cause obstruction, hemorrhage, and perforation frequently requiring surgery; discontinue medication immediately if abdominal pain, distention, nausea, vomiting or gastrointestinal bleeding occurs; Eutron does not contain added potassium. 7. Possible systemic lupus erythematosus has been reported for thiazides.

Precautions—Pargyline: Use cautiously at reduced dosage; caffeine, alcohol, antihistamines, barbiturates, chloral hydrate, other hypnotics, sedatives, tranquilizers, narcotics. Periodically do urinalyses, blood counts, liver function tests, etc. Use with caution in liver disease. Watch for orthostatic hypotension, especially in impaired circulation (e.g., angina pectoris, coronary artery disease, cerebral arteriosclerosis); also, augmented hypotension in concomitant febrile illnesses. Reduce or discontinue if hypotension is severe. In impaired renal function watch for cumulative drug effects, elevated BUN and other evidence of progressive renal failure; withdraw drug if these persist. In surgery increased central depressant response (hypotension and increased sedative effect) can be controlled by (1) discontinuing at least two weeks prior; (2) in emergency surgery lowering dose of premedication; (3) when necessary, administering a vasopressor. Do not use in hyperactive and hyperexcitable patients. Pargyline may unmask severe psychotic symptoms where emotional problems pre-exist. Use cautiously in Parkinsonism, especially with antiparkinsonian agents. In prolonged therapy, examine for change in color perception, visual fields, fundi and visual acuity. Also, prolonged therapy has made certain patients refractory to nerve blocking effects of local anesthetics.

Methyclothiazide: Use cautiously in severe renal dysfunction, impaired hepatic function or progressive liver disease; also in pregnancy (bone marrow depression, thrombocytopenia, and altered carbohydrate metabolism have been reported in certain newborn). In surgery thiazide may reduce vasopressor response and increase tubocurarine response. Antihypertensive response may be enhanced following sympathectomy. Watch for electrolyte imbalance (e.g., hyponatremia). Give supplemental chloride if hypochloremic alkalosis occurs and supplemental potassium if hypokalemia occurs (especially in digitalized patients). Thiazides may decrease serum P.B.I. without signs of thyroid disturbance.

Adverse Reactions — Pargyline: Orthostatic hypotension and associated symptoms, mild constipation, fluid retention, edema, dry mouth, sweating, increased appetite, arthralgia, nausea, vomiting, headache, insomnia, difficult in micturition, nightmares, impotence, delayed ejaculation, rash, purpura, weight gain, hyperexcitability, increased neuromuscular activity and other extrapyramidal symptoms. Drug fever is extremely rare. Reduction in blood sugar and hypoglycemic effects are possible. Congestive heart failure has been reported in a few patients with reduced cardiac reserve.

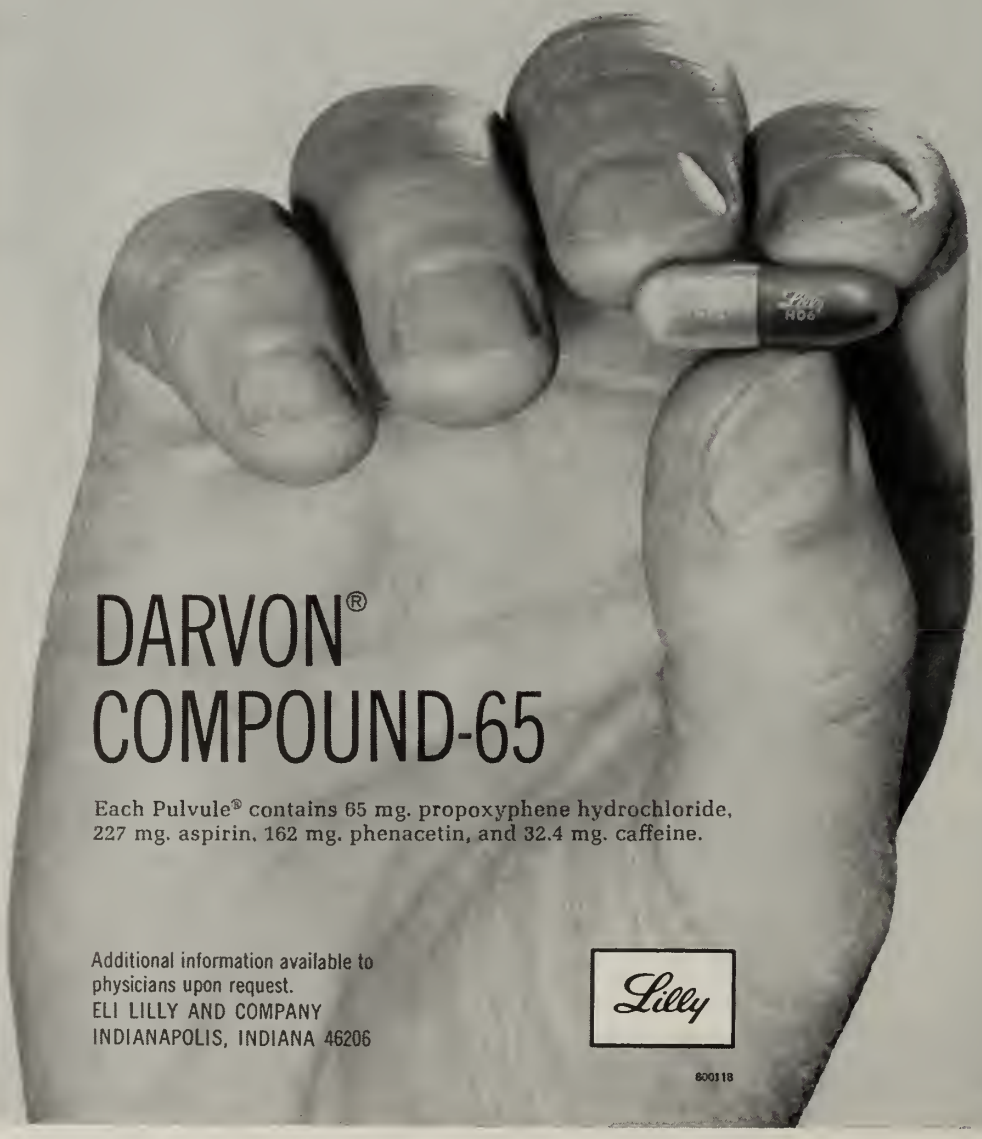
Methyclothiazide: Blood dyscrasias (thrombocytopenia with purpura, agranulocytosis, aplastic anemia); elevation of BUN, blood sugar or serum uric acid (gout may be induced); anorexia, nausea, vomiting, diarrhea, headache, dizziness, paresthesia, weakness, skin rash, photosensitivity, jaundice and pancreatitis. Cutaneous vasculitis in elderly patients has been reported with other thiazides.

If side effects are severe or persist, reduce dosage or withdraw drug. 804438R

ABBOTT

TM-TRADEMARK

Part of the fine art of medicine



DARVON® COMPOUND-65

Each Pulvule® contains 65 mg. propoxyphene hydrochloride, 227 mg. aspirin, 162 mg. phenacetin, and 32.4 mg. caffeine.

Additional information available to physicians upon request.
ELI LILLY AND COMPANY
INDIANAPOLIS, INDIANA 46206

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800118

The Virginia
MEDICAL MONTHLY

June, 1968
Vol. 95, No. 6
Whole No. 1393

Guest Editorial . . .

Support VaMPAC

1968 IS AN EXTREMELY IMPORTANT NATIONAL ELECTION YEAR. It is of utmost urgency for all Virginia physicians to become interested in and participate in political affairs which ultimately will affect each of us at the local level.

One of the ways active participation can be accomplished is by joining VaMPAC, the Virginia Medical Political Action Committee. This is a non-profit, non-partisan, unincorporated committee which utilizes education and active candidate support to aid political candidates whose philosophy is in harmony with organized medicine. The Board of Directors is appointed by The Medical Society of Virginia. VaMPAC is affiliated with the American Medical Political Action Committee nationally. Our main effort, however, is statewide and needs the backing of a majority of the membership of The Medical Society of Virginia.

Through financial contributions and active participation in public affairs we can provide the means for concerted political action. If we do not express our wants and needs about the current trend of government and legislation as relates to us, then we will not be properly represented. The membership of VaMPAC is composed of physicians, their wives, dentists, pharmacists and others. It provides a common meeting ground for exchange of ideas and active candidate support.

This Committee has accomplished much in the past and can accomplish much in the future if actively supported by the physicians of Virginia. A very real contribution was made by VaMPAC to the 1966 Congressional elections in Virginia and it has been said that we were *the* deciding factor in several campaigns. Join VaMPAC today by sending your voluntary contribution of $25.00 or more to Robert G. Stuart, Executive Secretary, VaMPAC, Post Office Box 2939, Richmond, Virginia 23235.

The survival of the free enterprise system as it relates to medicine depends on competent representation in Congress. Only if we are represented properly will our system survive. Our profession needs the active support of all its members today as never before.

Echols A. Hansbarger, Jr., M.D.

Lynchburg General Hospital
Lynchburg, Virginia 24501

A Clinical Review of Carcinoma of the Larynx

University of Virginia Hospital 1935-1964

T. H. HUTCHINSON, II, M.D.
Lakeland, Florida

J. B. GORMAN, M.D.
Lynchburg, Virginia

G. S. FITZ-HUGH, M.D.
Charlottesville, Virginia

This series is made up of 362 cases of epidermoid carcinoma of the larynx. The lack of improvement in the overall survival rate over the last 30 years emphasizes the need for better education of both physicians and the public concerning symptoms of laryngeal carcinoma as well as the need for early and vigorous treatment.

EPIDERMOID CARCINOMA of the larynx continues to be a formidable disease. This paper surveys all laryngeal carcinomas seen in the University of Virginia Hospital, Department of Otolaryngology, from 1935 to 1964. Cases are classified according to the T, N, M system (American General Committee for Cancer Studies and End Result Reporting) which divides the larynx into three anatomical sites: supraglottic, glottic and subglottic (see table I). This system immediately eliminates a large number of cases that were previously considered variations of laryngeal origin. These would include carcinomas of the pyriform sinus and the anterior, lingual surface of the epiglottis. All pyriform sinus carcinomas will be reported in a future publication.

Classification is based on clinical description gained by direct and/or indirect larnyngoscopy. However, when clinical description is inadequate, the data is supplemented by the pathologic description.

A total of 395 cases were studied. Thirty-three charts contained inadequate information for classification and are not included. The remaining 362 cases were divided into two time groups, one dating from 1935-1950, the other dating from 1951-1964. The indeterminates listed in these tables were subtracted from the total cases in determining the percentage of five and 10 year survivals (the indeterminates have lived less than five years after treatment or were lost to follow-up). Ten year survivals are included for interest because of the large number in the first time group.

Patients in the 1951-1964 (second time group) have had advantage of surgical pro-

HUTCHINSON, T. H. II, M.D., *Former Resident, Department of Otolaryngology, University of Virginia Hospital, now at Watson Clinic, Lakeland, Florida.*

GORMAN, J. B., M.D., *Lecturer, Department of Otolaryngology, University of Virginia Hospital.*

FITZ-HUGH, G. S., M.D., *Professor and Chairman of the Department of Otolaryngology, University of Virginia Hospital.*

Presented at annual meeting of Virginia Society of Ophthalmology and Otolaryngology, Hot Springs, April 1967.

TABLE I. TNM SYSTEM-LARYNGEAL CANCER CLASSIFIED BY ANATOMIC SITE OF ORIGIN

ANATOMIC DESCRIPTION

Supraglottic	*Glottic*	*Subglottic*
Posterior surface of epiglottis (including the tip) aryepiglottic fold, arytenoid, ventricular bands (false cords), ventricular cavities (right and left).	True vocal cords (right and left), anterior glottic commissure.	Subglottic region, exclusive of the under surface of true cords.

T_1—TUMOR CONFINED TO ONE ANATOMIC SITE WITHIN THE LARYNX

Tumor confined to laryngeal surface of epiglottis, or to an aryepiglottic fold or arytenoid, or to a ventricular cavity or a ventricular band.	Tumor confined to one vocal cord and mobility of cord remains normal.	Tumor limited to one side of the subglottic region, exclusive of the under surface of cord.

T_2—TUMOR CONFINED TO ONE ANATOMIC REGION WITHIN THE LARYNX

Tumor involving the epiglottis, extending to the ventricular cavities or bands, AE folds.	Tumor involving both cords with normal mobility of cords, or tumor of one or both cords with fixation of cord(s).	Tumor extending to two sides of subglottic region, exclusive of the under surface of cords.

T_3—TUMOR EXTENDING BEYOND ONE ANATOMIC REGION BUT CONFINED TO THE LARYNX

Tumor of the epiglottis and/or ventricles or ventricular bands, and extending onto the cords.	Tumor extending from cords either to subglottic region or supraglottic region, i.e., to ventricular bands or ventricles.	Tumor involving the subglottic region and extending onto the cords.

T_4—TUMOR EXTENDING BEYOND THE LARYNX

Tumors as in T_1, T_2, or T_3, but with direct extension to pyriform sinus, postcricoid region, vallecula, or base of tongue.	Tumors as in T_1, T_2, or T_3, but with direct extension through cartilage to skin, to the pyriform sinus, or to the postcricoid region.	Tumors as in T_1, T_2, or T_3, but with direct extension to trachea, skin or postcricoid region.

LYMPH NODE METASTASIS

N_0 No clinically palpable cervical lymph node(s) (metastasis not suspected).
N_1 Clinically palpable cervical lymph node(s) that are not fixed (metastasis suspected).
N_2 Clinically palpable lymph node(s) that are fixed (metastasis suspected).
M_0 No distant metastasis.
M_1 Clinical and/or radiographic evidence of metastasis except to the cervical lymph node regions.

CLINICAL STAGES

Stage I	T_1-N_0-M_0	Stage IV	T_4-N_1-M_0
Stage II	T_2-N_0-M_0		T_1-N_2-M_0
	T_3-N_0-M_0		T_2-N_2-M_0
	T_4-N_0-M_0		T_3-N_2-M_0
Stage III	T_1-N_1-M_0		T_4-N_2-M_0
	T_2-N_1-M_0		T_1-M_1
	T_3-N_1-M_0		

ADDENDUM: Additional information and copies of this classification may be obtained from:

The Executive Secretary
Joint Committee on Cancer Staging and End Result Reporting
40 East Erie Street
Chicago, Illinois

cedures such as neck dissection which was not frequently performed at the University Hospital prior to 1951. The large number of cases in stage II have been subdivided for clarity in certain tables.

Results

Only six of the 362 cases studied were classified as true subglottic tumors. These are not statistically significant and are not included in the tables. Four of these had laryngectomies alone and two had laryngectomies with neck dissection. There was only one five year survival (a laryngectomy), representing a survival rate of 16% for this group.

Table II shows the total number of patients increased from 97 in 1935-50 to 265 in 1951-64. During this same period, the

TABLE II. YEAR-GROUP DISTRIBUTION OF CASES

SITE	YEAR GROUP 1935-50	1951-64	Total
Supraglottic	30	79	109
Glottic	63	184	247
Subglottic	4	2	6
Total	97	265	362

TABLE III. SUPRAGLOTTIC GROUP DISTRIBUTION BY SEX

	1935-50			1951-64		
STAGE	Male	Female	% Dist.	Male	Female	% Dist.
I	4	1	17	19	3	27
II	16	3	63	24	7	39
III	3	0	10	13	2	19
IV	3	0	10	10	1	−15
Total	26	4	100	66	13	100

population in Virginia increased from 2,637,000 in 1935 to 4,456,000 in 1964.

In both time groups in table IV, the dominate decades were the sixth and seventh. In tables V, VI, IX and X the patients are listed according to their last surgical procedure.

Supraglottic

The supraglottic group as a whole contain 17 (16%) females (table IV), while the glottic group contained only 15 (6%) females (table VII.) The percentage of females in the supraglottic group grew from 12% in 1935-50 to 17% in 1951-64 (tables IV and VIII).

The 23 indeterminate patients in the 1951-64 supraglottic group (table VI) are all living less than five years following surgery. Two patients included under laryngectomy and neck dissection in stage II had subtotal laryngectomies. One is a five year survival and living, the other died of metastasis in less than five years. On stage IV patient underwent a subtotal laryngectomy and neck dissection following a pre-operative course of Cl_{60} and is alive three years after surgery with a proven second primary of the lung. In table VI, the 5-10 year survival for the stage II cases is only 20%. The $T_2N_0M_0$ cases is 36%. In contrast the $T_3N_0M_0$ patients had no five year survivals while the $T_4N_0M_0$ patients had only one. Altogether, they (T_3,T_4) represent about 50% of the total cases in stage II. This variance in results in the classification in stage II suggests that better guide lines may be needed to further classify this area. Interestingly, in table VI, the stage I patients having a laryngectomy and bilateral staged neck dissections had three five year survivals. Of the total of eight in this category, five were indeterminates who have not lived five years after surgery, although they are apparently free of disease at this time.

The over all five year survival rate of 19% reveals the gravity of supraglottic lesions and need for early diagnosis and vigorous therapy in this area. Based on the figures in table VI, laryngectomy and bilateral staged neck dissections appears to be the general treatment of choice. (The table shows a survival rate of 50% and eight indeterminates are still living). Total laryngectomy would not be the preferred treatment in certain cases, say those in which

supraglottic laryngectomy is possible, leaving a functioning larynx. However, staged bilateral neck dissection appears to be indicated in many cases, especially those located near or crossing the mid line.

has not increased significantly, since glottic symptoms usually appear earlier than supraglottic symptoms and thus may cause the patient to seek medical aid sooner. (Tables VIII, IX and X give additional data on the

Table IV. Supraglottic Group Age Distribution

	30-39	40-49	50-59	60-69	70-79	Total
			1935-50 Age Group			
Male	1 (3%)	5 (16%)	8 (28%)	8 (28%)	4 (13%)	26 (88%)
Female	0	1 (3%)	1 (3%)	1 (3%)	1 (3%)	4 (12%)
Total	1 (3%)	6 (19%)	9 (31%)	9 (31%)	5 (16%)	30 (100%)
			1951-64 Age Group			
Male	0	13 (17%)	27 (34%)	20 (25%)	6 (7%)	66 (83%)
Female	0	3 (4%)	4 (5%)	6 (8%)	0	13 (17%)
Total	0	16 (21%)	31 (39%)	26 (33%)	6 (7%)	79 (100%)

Table V. 1935-50 Supraglottic Group Treatment and Survival

	Stage							
	I	II*			III	IV		
		T_2	T_3	T_4				
	All Cases						Total	% 5-10 Yr. Survivals
Total patients	3	11	6	2	3	3	28	
Indeterminates	0	0	1	0	0	0	1	52
5-10-yr. survival	1	8	4	1	0	0	14	
	Laryngectomy							
Total patients	1	6	4	0	1	0	12	
Indeterminates	0	0	1	0	0	0	1	82
5-yr. survival	0	2	0	0	0	0	2	
10-yr. survival	0	4	3	0	0	0	7	
	Radiation							
Total patients	2	5	2	2	2	3	16	
Indeterminates	0	0	0	0	0	0	0	31
5-yr. survival	1	1	1	0	0	0	3	
10-yr. survival	0	1	0	1	0	0	2	

*(All were N_0 M_0.)

Glottic

As opposed to the findings in the supraglottic group (table III) the percentage distribution of the various stages in the glottic group are almost the same for the period of 1935-50 and 1951-64 (table VII). This may indicate that awareness of symptoms

glottic group). Two patients in the glottic group refused therapy following biopsy of their lesion. One of these is alive 15 years later without difficulty; apparently his lesion was removed at biopsy. The other patient expired of his disease. One patient included as a laryngectomy in table IX is

alive 25 years after radiation therapy and ten years after laryngectomy for a recurrence. A patient in the subtotal group underwent laryngectomy 13 years after his subtotal procedure and died of carcinomatosis two years after the second operation.

In table X, the 5-10 year survival ratio with various forms of therapy ranged from 38% to 80%. The subtotal group has an 80% five year survival, 20% higher than Cl_{60} irradiation today. Forty-eight of the 53 determinates in table X are alive but less than five years after treatment.

Several of the subtotal laryngectomies in stage I required further surgery. One had a laryngectomy and neck dissection six years after his subtotal procedure; he is alive one year after surgery. Two others underwent laryngectomy at two and four years respectively after subtotal laryngectomy. One is

TABLE VI. 1951-64 SUPRAGLOTTIC GROUP TREATMENT AND SURVIVAL

	Stage							
	I	II*			III	IV		
		T_2	T_3	T_4				
	All Cases						Total	% 5-20 Yr. Survivals
Total patients	21	13	8	11	14	12	79	
Indeterminates	7	2	1	4	2	7	23	19
5-10-yr. survival	4	4	0	1	2	0	11	
	Laryngectomy							
Total patients	3	3	3	3	2	2	17	
Indeterminates	0	0	0	1	1	2	4	15
5-yr. survival	0	1	0	0	0	0	1	
10-yr. survival	0	1	0	0	0	0	1	
	Laryngectomy and Radical Neck Dissection							
Total patients	8	5	3	4	6	4	30	
Indeterminates	1	1	0	1	1	2	6	17
5-yr. survival	1	2	0	0	1	0	4	
10-yr. survival	0	0	0	0	0	0	0	
	Laryngectomy and Bilateral Neck Dissection							
Total patients	8	2	1	2	3	1	17	
Indeterminates	5	1	1	1	0	1	9	50
5-yr. survival	3	0	0	0	1	0	4	
10-yr. survival	0	0	0	0	0	0	0	
	Radiation							
Total patients	2	2	1	2	3	5	15	
Indeterminates	1	0	0	1	0	2	4	9
5-yr. survival	0	0	0	1	0	0	1	
10-yr. survival	0	0	0	0	0	0	0	

*(All were N_0-M_0.)

those patients undergoing laryngectomy alone. Most of these subtotal laryngectomies were treated between 1951 and 1955 and based on their medical records, many of them probably would be candidates for alive four years after surgery. The other is alive at three years, apparently free of disease. Another underwent a second subtotal procedure and is living and well 12 years after his original operation. One patient in

stage II required laryngectomy two years after a subtotal procedure but did not survive five years.

Five radiation failures in stage II required surgery. One had a laryngectomy and neck dissection one year after therapy and is living three years following surgery, apparently free of disease. Another patient had a bilateral staged neck dissection and laryngectomy one year after radiation therapy and is alive five years after surgery. Another patient underwent a subtotal procedure five years following radiation therapy and is alive and well two years following surgery. Two others, having laryngectomy two years after radiation therapy, died with disease in less than two years.

Two stage I patients, first treated by radiation, eventually came to surgery. One had a subtotal laryngectomy six months after radiation and is living and well seven months post-operatively. The other had a subtotal procedure one year following Co_{60} radiation and is alive and well three years after surgery. One stage II patient, irradiation failure, refused surgery for three years. After receiving two courses of Co_{60} (a total of 10,000 r) he finally agreed to undergo laryngectomy and neck dissection but died less than two years later with generalized metastasis. There are four patients not included in table IX who refused therapy and were lost to follow up. Two were stage III and two were stage IV.

Table VII

Glottic Group Distribution by Sex

Stage	1935-50 Male	1935-50 Female	1935-50 % Dist.	1951-64 Male	1951-64 Female	1951-64 % Dist.
I						
T_1	26	0	41	72	7	42
II						
T_2	16	3		35	2	
T_3	10	2	49	51	1	48
T_4	0	0		1	0	
III	5	0	8	11	0	8
IV	1	0	2	4	0	2
Total	58	5	100	174	10	100

Table VIII

Glottic Group Age Distribution

	1935-50 Age Group						
	30-39	40-49	50-59	60-69	70-79	80-89	Total
Male	2 (3%)	9 (14.5%)	20 (32%)	14 (22%)	11 (18%)	2 (3%)	58 (92.5%)
Female	0	1 (1.5%)	2 (3%)	2 (3%)	0	0	5 (7.5%)
	2 (3%)	10 (16%)	22 (35%)	16 (25%)	11 (18%)	2 (3%)	63 (100%)
	1951-64 Age Group						
Male	8 (4%)	16 (8.5%)	51 (27.5%)	64 (35.5%)	26 (14.5%)	9 (4.5%)	174 (94%)
Female	0	1 (0.5%)	6 (3.5%)	1 (0.5%)	1 (0.5%)	1 (0.5%)	10 (6%)
	8 (4%)	17 (9%)	57 (31%)	65 (36%)	27 (15%)	10 (5%)	184 (100%)

Summary of Results

1. Three hundred and sixty-two cases of epidermoid carcinoma of the larynx are classified, 109 supraglottic, 247 glottic and six subglottic. The latter group was not considered statistically significant.

2. The dominant decades in both the supraglottic and glottic groups were the sixth and seventh.

3. The thirty year 5-10 year survival rate was about 31% for the supraglottic group and 63% for the glottic group.

4. The supraglottic survivals varied from 52% in the first time group to 19% in the second time group. The reasons for this drop is not clear. The large number of living indeterminates, less than five years after surgery, in the second time group should improve this survival percentage.

5. The survival rates between the two time groups in the glottic group are not appreciably different (67% versus 60%).

6. Large numbers of living indeterminates in the second time glottic group (table X) can be expected to increase the five year survival rate. A large percentage of these are alive less than five years after surgery.

7. In table X, the patients undergoing subtotal laryngectomy have the highest survival rate (80%).

Table IX

1935-50 Glottic Group Treatment and Survival

	Stage							
	I	II*			III	IV		
		T_2	T_3	T_4				
	All Cases						Total	% 5-10-Yr. Survivals
Total patients..	26	19	12	0	5	1	63	
Indeterminates..	1	0	2	0	1	1	5	67
5-10-yr. survival.	17	11	9	0	1	0	38	
	Laryngectomy							
Total patients..	8	9	10	0	3	1	31	
Indeterminates..	0	0	1	0	1	1	3	65
5-yr. survival..	1	1	1	0	1	0	4	
10-yr. survival.	3	3	8	0	0	0	14	
	Subtotal Laryngectomy							
Total patients..	16	8	1	0	1	0	26	
Indeterminates..	1	0	0	0	0	0	1	76
5-yr. survival..	4	1	0	0	1	0	6	
10-yr. survival..	8	5	0	0	0	0	13	
	Radiation							
Total patients..	2	2	1	0	1	0	6	
Indeterminates..	0	0	1	0	0	0	1	40
5-yr. survival..	1	1	0	0	0	0	2	
10-yr. survival..	0	0	0	0	0	0	0	

*(All were N_0-M_0.)

Discussion

Many difficulties were encountered in attempting to determine the 5-10 year survival in this group of patients. The University of Virginia Hospital is in a rural area and the majority of patients come from locations up to several hundred miles away. This makes difficult both follow up of those who live and determination of the cause of death on those who die outside the immediate vicinity. Information on the actual cause of death often is not available, therefore, all deaths in this study were presumed to be from carcinoma when there was no

information to the contrary. However, the sixth and seventh decades are predominate in both the supraglottic and glottic groups and it is apparent that many patients might have died from other diseases.

sified campaigns waged against cancer in recent years, although it is known that the incidence of cancer is generally on the rise. The increase in the population of the state also is a contributing factor.

TABLE X

1951-64 GLOTTIC GROUP TREATMENT AND SURVIVAL

	Stage							
	I	II*			III	IV		
		T_2	T_3	T_4				
	All Cases						Total	% 5-10-Yr. Survivals
Total patients...	78	37	50	1	13	5	184	
Indeterminates..	18	15	15	0	4	1	53	60
5-10-yr. survival.	41	15	20	0	2	1	79	
	Laryngectomy							
Total patients...	11	14	13	0	0	1	39	
Indeterminates..	2	2	4	0	0	0	8	60
5-yr. survival..	3	2	4	0	0	1	10	
10-yr. survival..	1	5	2	0	0	0	8	
	Laryngectomy and Radical Neck Dissection							
Total patients...	1	9	26	1	6	2	45	
Indeterminates..	0	6	9	0	2	1	18	48
5-yr. survival..	1	0	9	0	1	0	11	
10-yr. survival..	0	2	0	0	0	0	2	
	Laryngectomy and Bilateral Neck Dissection							
Total patients...	1	2	0	0	4	2	9	
Indeterminates..	0	1	0	0	0	0	1	38
5-yr. survival..	1	1	0	0	1	0	3	
10-yr. survival..	0	0	0	0	0	0	0	
	Subtotal Laryngectomy							
Total patients...	37	7	4	0	2	0	50	
Indeterminates..	4	3	1	0	1	0	9	80
5-yr. survival..	17	3	3	0	0	0	22	
10-yr. survival..	9	1	1	0	0	0	11	
	Radiation							
Total patients...	28	5	7	0	1	0	41	
Indeterminates..	12	3	1	0	1	1	17	50
5-yr. survival..	7	1	2	0	0	0	10	
10-yr. survival..	2	0	0	0	0	0	2	

*(All were N_0-M_0.)

The large increase in the number of patients treated in the 1951-64 group may be partly accounted for by the fact that many patients who are operated on today would have been considered inoperable 15 or 20 years ago. Another factor may be the intensified campaigns waged against cancer in recent years, although it is known that the incidence of cancer is generally on the rise. The increase in the population of the state also is a contributing factor.

There is no ready explanation for the lack of improvement in survival rate in the past 15 years but large numbers of living indeterminates should improve the survival in both groups.

The survival rate for patients with exten-

sive lesions (stages III and IV) is not much improved, even when including the indeterminates. This points out the gravity of extensive lesions, the need for earlier diagnosis and more vigorous therapy, and the possible combination of surgery, irradiation and antimetabolites. At the University of Virginia Hospital, patients with extensive lesions in this area now are receiving preoperative irradiation. Our early clinical impression is that this treatment will increase the number of long term survivals.

Summary

A clinical study of 362 epidermoid carcinomas of the larynx treated at the University of Virginia Hospital between 1935 and 1964 indicates that this is still a formidable disease. Patients in the 1935-50 group had a higher over all survival rate (63%) than those in the 1951-64 group (50%). A large number of living indeterminates in the second time group is expected to increase this survival percentage. A combination of surgery and pre-operative irradiation is now being used on extensive lesions and is expected to increase the number of five year survivals.

Results emphasize the need for better education of both physicians and the public concerning symptoms of laryngeal carcinoma and the need for early otolaryngic examination and diagnosis.

University of Virginia Hospital
Charlottesville, Virginia 22901

MONTHLY REPORT OF BUREAU OF COMMUNICABLE DISEASE CONTROL

	Apr. 1968	Apr. 1967	Jan.-Apr. 1968	Jan.-Apr. 1967
Brucellosis	9	1	11	12
Diphtheria	0	0	0	0
Hepatitis	43	103	169	265
Meningitis (Aseptic)	1	0	1	3
Meningococcal Infections	4	3	19	15
Poliomyelitis	0	0	0	0
Rocky Mt. Spotted Fever	3	1	5	1
Rubella	121	150	343	293
Rubeola	50	577	189	1417
Streptococcal Sore Throat (including Scarlet Fever)	1236	1716	6401	6641
Tularemia	0	0	1	0
Typhoid Fever	0	0	3	2
Rabies (in animals)	10	46	71	107
Venereal Diseases				
Syphilis	159	197	539	732
Gonorrhea	674	743	3020	2886
Other	2	3	5	4

Benign Mucous Membrane Pemphigoid

JAMES PARKER CROSS, JR., M.D.
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The course of benign mucous membrane pemphigoid over a six year period in a colored female is described. The literature is reviewed and the disease discussed.

THIS PAPER describes the course of benign mucous membrane pemphigoid in a 36 year old colored female whose history is known in various degrees of certainty over a six year period.

The first case of esophageal pemphigoid with scarring was reported by Adam[2] in 1910 who attempted a blind bougienage which caused death three days postoperatively with a high fever. The upper portion of the esophagus and larynx at necropsy showed severe scarring. In 1917, Thost[3] described a man who died of pneumonia at age 82 who apparently had had pemphigoid since the age of 50. Multiple esophageal webs were noted at autopsy. In 1933[4] Baumann described a "deeply congested epiglottis with blebs" and esophageal stenosis.

In 1925, New[6] reported on nineteen cases, eleven of which involved the larynx. The ages were from 20 to 76. The disease had been present for two weeks prior to being seen, with the average duration of the disease being 18½ months. The mouth was affected in 19 patients; pharynx, 14; larynx, 11; esophagus, 1; nasopharynx, 1; and nose, 1. Five had no involvement of the skin. Four had vaginal and/or rectal mucosal involvement. Eight had conjunctival involvement. Clinically, the disease was characterized by the development of from one to several bullae, which ruptured in an unpredictable time causing the formation of a superficial membrane. Usually the membrane healed without residua, but extensive ulceration occasionally did occur. No scarring was usually noted in the mouth but if the disease involved the larynx, eventual cicatricial obstruction was frequently found.

From his experience with four patients, Benedict[1] presented several comments of interest in 1952. He felt the disease not to be related to pemphigus foliaceous, pemphigus vegetans, or pemphigus vulgaris, either in its clinical course or its histological picture. Pemphigoid gave rise to scarring which did not occur in pemphigus. Fifty per cent of his cases had skin lesions along with mucosal bullae and erosions. The skin lesion were of two types, one of which was that of discrete bullae which healed without scarring; the other of which was that of recurrent bullae with resultant smooth atrophy of the involved skin.

He noted that the conjunctivae were the most frequently involved sites, but that the disease could occur without any eye involvement at all. Corneal scarring with blindness was described.

After conjunctival disease, the order of involvement was the mucosa of the mouth and oropharynx, nose, larynx, esophagus, glans penis, vulvar and anal mucosa.

Histologically, Benedict described subepidermal bullae with normal overlying epidermis. (In true pemphigus there is acantholysis, apparently.[5]) Jamieson[7] 1962, felt that benign mucous membrane pemphigoid differed from true pemphigus and that other vesicular lesions which had to be considered

Presented at annual meeting of Virginia Society of Ophthalmology and Otolaryngology, Hot Springs, April 1967.

in the differential diagnosis were: 1, bullous lichen planus; 2, erythema multiforme bullosum; 3, herpetic gingivostomatitis; 4, aphthous stomatitis; 5, Reiter's disease. In agreement with other earlier authors, he found no incidence of the patient's general health being primarily affected.

Wallner[5] reported on five patients in 1964. He used the term "boiled milk" membrane with ulceration in describing the oral mucosa appearance and suggested either that this membrane did not always go through the bleb stage, or that blebs were very difficult to find as they might appear and rupture within a very short period.

In his series there were no deaths noted, the patients being treated with very high doses of Prednisone, even as much as 40 5 mg. tablets per day. Eventual reduction to a maintenance level sufficient to prevent formation of new lesions was his goal. There is no other known effective treatment.

From Wallner's review of the problem and his personal experience, he suggested that perhaps the problem was not so rare as previously thought.

History

In November, 1960, the patient, a colored female, then 30 years old, was admitted to the Norfolk General Hospital clinic service for evaluation of blood-tinged sputum with "flecks of tissue" in it. White blisters were described on the left tonsil and skin lesions were noted. After a dermatology consultation, she was given steroid therapy with relief.

She was then lost to follow-up until four years later, August, 1964, at which time she was seen in my office because of dysphagia, odonophagia, and weight loss. The dysphagia had been minimally progressive for two years until two months prior to being seen by me, when her symptoms became markedly worse. She described recurrent mouth pain, bleeding and oral ulcerations, a foul taste, and bad breath.

Physical examination revealed moderate nasal mucosa crusting and a normal nasopharynx. All over the mucosa within the mouth and oropharynx was a more or less continuous "boiled milk" membrane with shallow ulcerations. The epithelium bled easily even with the most gentle touch, being avulsed from the underlying tissue. Similar lesions were present on the epiglottis and about the esophageal introitus. The vocal cords were normal.

Four months before we saw her, the referring physican had obtained an esophagogram with fluoroscopy (i.e. two months prior to her increased dysphagia). This was read as normal, but subsequent films indicated that probably an area of cervical esophageal narrowing was present even then in April, 1964. Her first esophagogram with us in August, 1964, revealed dilation of the right antero-lateral pyriformis with delayed barium bolus passage at a gradual constriction involving the C7-D1 area, and a web at C6.

Attempts at local anesthesia esophagoscopy were met with failure because of pain in spite of topical cocaine, causing (or concurrent with) much cricopharyngeus spasm. General anesthesia was then supplied and an 8mm. esophagoscope introduced. There was a web noted anteriorly just within the cricopharyngeal pinchcock which was easily avulsed with minimal bleeding. In this area the mucosa was somewhat friable. Moderate tightness was noted at the C7-D2 area. The remainder of the esophagus was normal.

Six weeks afterwards she had been swallowing well since almost immediately after the esophagoscopy and had gained 17 pounds. In four more months, she had gained a total of 35 pounds. She was on steroid therapy during the first five months after initial endoscopy.

Unfortunately, she was again lost to follow-up for two years, and when she returned, it was because of two weeks' dysphagia with a painful mouth. She had not been on any consistent steroid treatment. A barium swallow with cine revealed

the entire hypopharynx to be diminished in size, the epiglottis to be horizontally displaced and thickened, and a persistent narrowing at C6-7. Pyriformis filling was minimal and the valleculae were shrunken. In the distal esophagus, there was a tapered narrowing with absence of the usual longitudinal mucosal folds. Swallowing initiation was difficult.

An attempted laryngoscopy and esophagoscopy under good general anesthesia was aborted because the epiglottis was so rigid and horizontally displaced that its elevation with the tip of the laryngoscope caused a horizontal rotation forward of the larynx in a fixed manner so as to preclude glottic visualization. It was not felt to be safe to proceed. However, since we knew the diagnosis and had good contrast studies we performed Bougienage with Maloney dilators, through # 40 size.

Swallowing improvement was immediate and a follow-up barium swallow revealed easier deglutition, the elimination of esophagogastric spasm and the institution of normal peristalsis in the area, but with persistent narrowing. She was again placed on steroids which she intermittently took, but three months later (in my absence) she was readmitted with marked airway obstruction. A tracheotomy was anticipated but her acute problems diminished with steam inhalation. The epiglottis was easily visible without a mirror and she complained of a lump sensation in that area. It is felt that this thick horizontal epiglottis was the cause of her obstruction. Laryngograms revealed good false and true cord motion without laryngeal obstruction per se, but the epiglottis rested on the posterior hypopharynx. Space between the epiglottis and posterior wall was only noted when artificially created by a Valsalva maneuver. She was again dilated through a # 40 Maloney bougie, and placed on 80 mg. of Prednisone daily (February, 1967). This was gradually reduced to 60 mg., and she has stayed on this dosage up to the present time. In spite of this she still develops an occasional new lesion, but eats and swallows well, and her airway is good.

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Hip Problems in the Paraplegic Patient

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After proper rehabilitation, many paraplegics will lead useful and successful lives. Orthopedic treatment of hip disorders is an important part of this rehabilitation. Adductor tenotomy and resection of the head and neck of the femur are among procedures used.

ONE MIGHT SAY that discussion of any subject is best handled by jotting down one's own thoughts and observations as they have been influenced by what others have written and talked about on the subject. This I have done, and for this thanks must go to Dr. Phillip Oden, Chief of Spinal Cord Injury Service at the Veterans Hospital in Richmond, and the various Orthopedic men on the staff of the Veterans Hospital who have discussed each and every paraplegic case which I felt might be benefited by orthopedic evaluation and possible surgery during the past seven years of work at the Veterans Hospital. During this period, it has been my privilege to see in consultation all new quadriplegic and paraplegic admissions to determine if I felt that they needed orthopedic work-up and possible surgery, or other corrective measures.

With the paraplegic patient, the basic procedure is the same as with any other patient: First, you must determine your goal to be achieved; Second, obtain complete cooperation of the patient; and last, determine the methods best suited in reaching your goal. In applying this basic procedure to the paraplegic, however, two problems *peculiar* to this type of patient must be realized:

1. First, the paraplegic patient as an individual is chiefly concerned in knowing exactly what capacity for *ambulation* he has and what can possibly be obtained. (In reviewing the minutes of the Spinal Cord Injury Conferences, I learned that Dr. Ernest Bors of the VA Hospital in Long Beach, California, and Dr. Estin Comarr of West Roxbury in Boston both concur in this consideration.) Because of this concern on the part of the paraplegic, he must be made to know and understand that he will either be a *walker* or a *sitter*. As a *walker*,



Slide #1

(slide #1) which is the best result that can be obtained, he must have braces and full

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hip extension with passive flexion and crutches. The paraplegics who become "walkers" are usually younger and learn quite readily to walk with a swing through gait.

As a *sitter* (slide #2) the paraplegic will



Slide #2

be a full time wheelchair patient with flexion and extension of the hips. All pain and spasm must be taken care of and the patient must be able to sit straight and comfortably all day in his wheelchair. This particular patient has attempted standing, but because of decubitus ulcerations on the heels, he has sat most of the time.

2. The second problem peculiar in paraplegic treatment is that surgical intervention must be helpful and *usually of a relatively simple nature*. In spite of the fact that often times there is no motor or sensory perception present in the limbs, paraplegic patients are *particularly* apprehensive about losing their extremities, and special effort to avoid amputation on this type of patient should be made. Also, the paraplegic patient is especially prone to infection and decubitus ulcers. For these reasons, *special* care must be taken to have the end result of surgical procedures beneficial rather than harmful. It is certainly true that fractures do heal, but it is just as true that soft tissues of the paraplegic are fraught with disaster.

Example: (slide #3) This patient was



Slide #3

a paraplegic who developed severe myositis ossificans bilaterally with complete stiffness of the hips. Hip surgery with local excision was performed. He ended up with severe infection and had disarticulation of both hips. Today, he is a bilateral *amputee*, whereas before he had both extremities with complete fixation at both hips. I think that one must conclude that the surgery was definitely not beneficial. In retrospect, this patient was a long term paraplegic with soft tissues too poor for treatment by surgery.

Having discussed two *general* problem areas peculiar in the treatment of paraplegics, let me now discuss a problem of a more specific nature—that of hips. At the Veterans Hospital in Richmond, there are large numbers of paraplegics as a result of Vietnam casualties, gunshot wounds, and automobile accidents who need hip reconstruction. Surgery of the hips in the paraplegic patient is basically concerned with attempting to achieve painless motion. (slide #2)

One method of surgery which we have used to achieve this end is *adductor tenotomy*. This procedure entails cutting the pelvic attachments of the adductor magnus, longus and brevis, and/or parts of each of these. The psoas tendon at its lesser trochanter attachment may also be a deforming factor. This patient had scissoring and after adductor tenotomy wears a brace to keep his knees from returning to their original position, as seen in this slide.

A second method is that of resection of the head and neck of the femur which is done by making either a transverse femoral incision and extracting the head and neck, or by making a regular hip nailing incision. We have used both methods and find that the condition of the skin over the greater trochanter is the determining factor. Our good results were obtained if there was good surrounding muscle to fill in the dead space where the femur was removed. Eight cases in the past three years have shown this to be an excellent procedure if no secondary infection occurs and the wounds heal per primam. Use of a hemovac is helpful and is employed routinely.

At the Veterans Hospital in Richmond, the most *frequent* hip problem is that of



Slide #4

chronic draining sinus secondary to decubiti and with or without osteomyelitis and dislocation of the hips secondary to infection. Work-up of these patients includes a sinogram, surgical correction, and also plastic surgery to reconstruct tissue coverage which is done by our plastic surgeon, Dr. Hunter Jackson. A sinogram (slide #4) is done in the x-ray department by placing the patient on the x-ray table with the side for injection up. Five to ten ccs. of hypaque are used with a small sterile plastic tube similar to that found in an intracath. Lateral, oblique, and anterior-posterior x-rays are taken to determine the extent of the sinus tract. As shown here, the extent of the infected tract is limited to the greater trochanter and does not extend into the joint. For this reason, it is not necessary to resect the head of the hip bone. In more severe cases, the head of the femur may be dislocated, and the joint must be removed. This particular patient, a 30 year old colored male, had resection of the head and neck of the femur since he had marked osteomyelitis. Following surgery, his draining sinus healed completely.

This x-ray (slide #5) is of the same pa-



Slide #5

tient, a year after surgery. He has flexion of the hip to 90 degrees, but as one can plainly see, there is reformation of bone and over the next few years, he may form some stiffness in the hips, and need further treatment.

As we can see from this case, resection of the head and neck of the femur was successful, but often times, the results are dis-

couraging since patients sometimes tend to develop infection in the local site due to the dead space from bone removal, and will eventually come to ablation of the extremity as in this case. (slide #3) For this reason, each case must be studied individually to determine the best method, always keeping in mind that soft tissues of the paraplegic are fraught with disaster.

In summary, I should simply like to say that the paraplegic patient is ever with us in this industrial and competitive age. Our plan must be to concern ourselves with each one *individually*, to determine the method of treatment according to individual need, and to ambulate and rehabilitate each one according to his own capacity and ability. Hips must be corrected to a functional position as best for that particular patient; then, we must follow each one in ensuing years to prevent contractures, infection, and decubiti. If this is done, many will lead useful and successful lives.

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Arthrogryposis Multiplex Congenita Associated with Von Recklinghausen's Disease

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A case of arthrogryposis multiplex congenita associated with Von Recklinghausen's neurofibromatosis is reported. Clinical and autopsy findings are discussed. This is a most unusual and interesting case.

SINCE the original report by Otto[1] in 1841 there have appeared in literature other cases of arthrogryposis multiplex congenita, but we have failed to discover the definite proof of an association of this neuromuscular and skeletal disorder with Von Recklinghausen's disease (multiple neurofibromatosis). It is the primary intent of this paper to describe the association of these two conditions.

Review of Literature

The standard texts of neurology do not give a complete picture of arthrogryposis, and most of the facts were obtained from case reports. Relkin[2] recently reported a case of Von Recklinghausen's disease in a sixty-three-year-old female who is still alive and also has arthrogryposis multiplex congenita. This patient had been admitted with the diagnosis of Von Recklinghausen's disease. However, on histological examination of a skin nodule an epidermal inclusion cyst was found. Other authors report the coexistence of arthrogryposis multiplex with congenital aortic stenosis,[3] with Pierre Robin syndrome,[4] with hyperostosis frontales interna,[5] with unilateral facial paralysis,[6] with Marfan's syndrome,[7] Ullrich syndrome,[8] Moebius' syndrome,[9] Klippel-Feil syndrome, syndactylism, spina bifida, cerebral palsy, craniostosis,[10] osteogenesis imperfecta,[11] and with tuberous sclerosis.[12]

The clinical aspects of this case should be considered in the light of the rubella syndrome, also, to include manifestations which were not recognized as rubella associated defects prior to the epidemic in 1964 (Cooper and Krugman).[13]

Other synonyms describing arthrogryposis multiplex congenita are myodystrophia foetales deformans,[14] congenital arthromyodysplasia,[15] multiple congenital rigidities,[16] and amyoplasia congenita.[17]

In none of the cases reported were any chromosomal studies performed.

Case Report

The child was sixteen months old when he was committed to the Central State Hospital on September 22, 1960, as a mental

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defective. The reason given for commitment was that the child had a disease of the peripheral nervous system known as neurofibromatosis, hydrocephalus, and multiple deformities of both upper and lower extremities. The personal history states that the boy was delivered by forceps in a hospital on May 31, 1959, after a prolonged labor. His birth weight was 7 lbs. 14 oz. The mother stated that the child was never trainable and that he constantly pushed his fingers into his left eye and this caused it to be markedly irritated. During the night the patient would awake and moan and groan, but no one was able to understand what he was trying to say.

On hospital admission the child was unable to sit up. His weight was 37 lbs. and height 33 ins. His heart was normal in size, shape and position with regular sinus rhythm and no murmurs. Lungs were clear, resonant and no rales were heard. There was asymmetry of the head with marked prominence of the right fronto-parietal region and marked dolichocephaly with widely open anterior fontanelle. The face was asymmetric with bilateral marked proptosis and ectropion of both palpebrae on the right. The jaw was small with high arched palate. There were translucent corneas bilaterally and extremely unequal miotic pupils. There was bilateral glaucoma and bilateral coarse horizontal nystagmus on conjugate lateral gaze. Widespread areas of increased pigmentation (cafe au lait) were seen involving the right side of the forehead, and the right side of the trunk and thigh posteriorly. There were deformities in both the upper and lower extremities with fixity of the joints, hyperactive reflexes more marked on the left (Fig. 1). Noticeable was the increased size of the abdomen and the enlargement of the affected limb muscles and joints, due to the excessive amount of fat. There also was lack of development of muscle mass and deformities of the hips, knees and ankle joints.



Fig. 1. Showing the deformities and sausage-shaped appearance of the extremities, hydrocephalus, the café au lait spots on the abdomen and thigh and the ptosis of the eyelids.

The psychological examination indicated that the child was mentally deficient to a severe degree and could not talk.

Laboratory Findings:

Laboratory studies. Hemoglobin 11.2 mgs.%; white blood cell count and differentiation were within normal range. The VDRL was non-reactive. Blood urea nitrogen, fasting blood glucose, serum proteins, calcium and phosphorus were all within normal limits. The cerebrospinal fluid was unremarkable.

Roentgen Studies: X-ray of chest showed marked obesity. The osseous structures were not remarkable. The lung fields were clear. The AP projection showed findings suggestive of amyoplasia congenita in that the subcutaneous fatty tissue appeared to be more abundant than would be expected with the relative quantity of soft tissue present.

Routine views of the skull revealed a definite dolichocephalic configuration as well as considerable asymmetry in the frontal views. The cranial bones were thin. The sutures were still open and the posterior fontanelle large.

A lateral projection of the lumber spine showed a scoliosis centered at the dorsolum bar area.

X-rays of the pelvis and lower extremities showed that there was dislocation of the right hip with flattening and irregularity of the acetabulum on the right. There was an old healed fracture in the proximal end of the left femur just below the level of the lesser trochanter (Fig. 2). The right



Fig. 2. There is dislocation of the right hip with flattening and irregularity of the acetabulum on the right. There is an old healed fracture in the proximal end of the left femur just below the level of the lesser trochanter.

femur also showed an old healed fracture just above the condyles. The right tibia showed fairly marked bowing anteriorly near the mid portion. There was marked dorsal flexion of the right foot with resulting compression deformity of most of the tarsal bones. The left foot showed marked plantar flexion.

Electroencephalogram: EEG showed generalized slow activity especially over the left hemisphere. The right hemisphere activity appeared generally depressed and more irregular. There were also some seizure discharges in the right parietal area.

Surgical Pathology Report of a Skin Nodule from Scalp: Normal skin and a neurofibroma.

Chromosomal Studies. Chromosome count: there were 46 chromosomes. No abnormality was seen in either the number or the morphology.

Hospital Course: The child remained bedridden. He could not move, sit or talk. He had to be spoon fed and required custodial care all the time.

On June 26, 1964, the patient was treated for a displaced fracture of the upper left femur and a plaster case was applied. On August 19, 1964, the cast was removed and the child made an uneventful recovery. On October 21, 1965, the child developed a temperature of 103.2° with crackling rales and harsh breath sounds at the right base. The rest of the physical examination was negative except for his pre-existing condition. A catheterized urine specimen revealed innumerable white blood cells, a few red blood cells and a 3 plus albumin. His white blood cell count was 16,000 with a shift to the left. A diagnosis of pneumonitis and urinary infection was made and the child was treated with parenteral fluids and antibiotics. His urinary findings and his white blood cell count gradually returned to normal.

On February 8, 1966, the child developed a temperature of 104.6°, pulse 136 and respiration 28. There were bilateral harsh breath sounds and crackling rales at the bases. A catherized specimen of urine revealed a 3 plus albumin and the microscopic field was loaded with both white and red blood cells. A mass pressing on the urinary bladder the size of a small grapefruit was palpable above the symphysis. The patient was given supportive treatment and antibiotics and placed in an oxygen tent. His condition continued to deteriorate and in spite of further therapy he became moribund and expired February 26, 1966.

Autopsy: Essential findings on external examination were "cafe au lait" spots on the skin of neck and upper chest, an enlarged dolichocephalic skull with multiple small nodules in the scalp, and multiple skeletal deformities. These deformities consisted of a small jaw, kypho-scoliosis of vertebral-column, complete fixation or severe restriction in motion of all joints, dislocation of right hip, healed fractures of both femoral bones, flexion deformity of both wrists

with formation of claw hands, calcaneovarus fixation of right foot. There was general atrophy of skeletal musculature but the right calf showed sausage-shaped outlines with marked increase in volume. The penis was of adult proportions but there was cryptorchism.

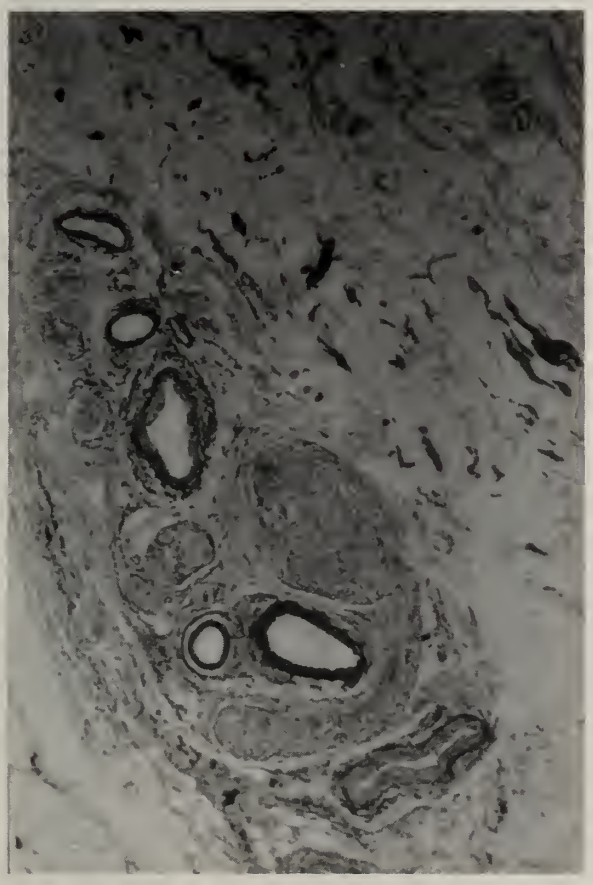


Fig. 3. Microscopic: Cross section of musculature of calf showing marked muscular atrophy and extensive adipose tissue replacement. Note increase in volume of two nerve trunks due to neurolemmal proliferation. (Masson trichrome, 12x)

On incision the subcutaneous fat was markedly reduced in amount. Internal examination revealed the abdominal muculature to be thin although it was of normal consistency and color as were both diaphragms. The organs within the thoracic and peritoneal cavities were grossly unremarkable. The left retroperitoneal space, however, showed two variably sized, ovoid tumors, the larger one extending into the pelvis and causing compression of the left ureter resulting in left hydroureter and hydroenphrosis. There was evidence of chronic urinary infection. The cranial cavity revealed a hydrocephalus involving both hemispheres and obstruction of the aqueductus Sylvii close to the third ventricle was found. The brain stem, cerebellum and cervical cord were grossly unremarkable.

Microscopic examinations confirmed a chronic urinary tract infection. In addition, focal necrosis of the liver was suggestive of septicemia and focal hemorrhagic changes in both adrenals were explained best on the same basis which was considered the immediate cause of death. The most striking findings as to the musculo-skeletal system were seen in a section from the pseudohypertrophic, right calf showing marked muscular atrophy and extensive adipose tissue replacement. Nerve trunks showed neurolemmal proliferation. (Fig. 3) The joint cartilage of the right ankle appeared to be unremarkable. Microscopic findings of the brain were in keeping with a hydrocephalus and gliotic stenosis of the anterior aqueductus Sylvii as known to occur in neurofibromatosis. Section of the cervical spinal cord showed diminution in number and shrinkage of nerve cells particularly in the anterior horns. However, there was no evidence of inflammation. Sections of the retroperitoneal tumors revealed fibro-collagenous tissue of varying density in which whorled bundles or interlacing fibrils of slender cells resembling Schwann cells (neurilemma) were embedded in haphazard fashion with considerable variety of the histologic pattern. There was also a suggestion of palisading in a few areas. A few questionable ganglion cells were seen but special stain (Nissl) did not confirm their presence. Hyperplasia of neurilemma and perineural connective tissue in varying arrangements were also found in multiple other locations as

in the kidneys (peripelvic) (Fig. 4), adrenals (pericortical), urinary bladder (intramural) (Fig. 5), within the ankle joint

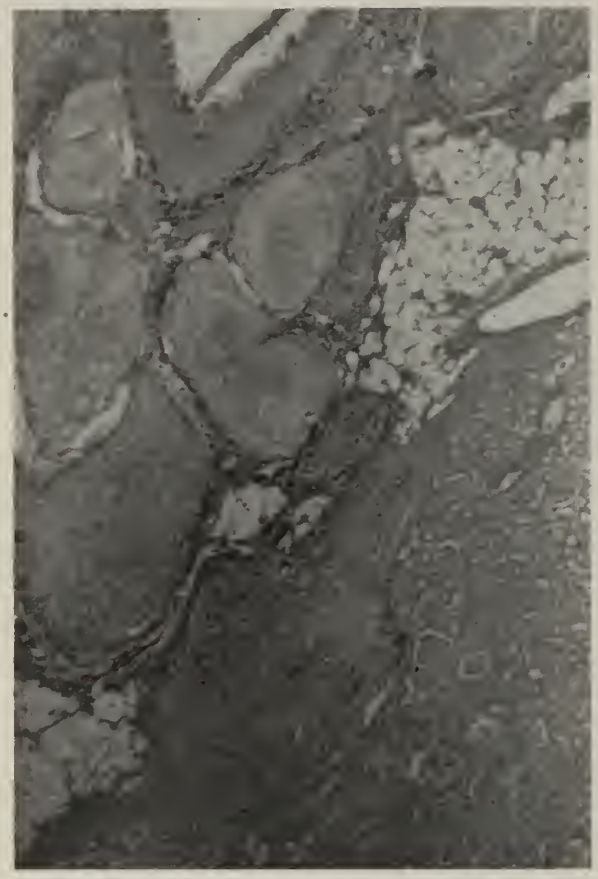


Fig. 4. Microscopic: Kidney displaying hyperplasia of neurilemma and perineural connective tissue. Note the partially club shaped nerve trunks occupying almost one half of the picture. (H & E, 16x)

space and underneath the skin. Sections of the nodes underneath the scalp were reported as neurofibroma during patient's previous hospitalization and were not repeated on autopsy.

Discussion

There is no doubt that the clinical and pathological findings in this case qualify for the diagnoses of both arthrogryposis multiplex congenita and generalized neurofibromatosis.

Arthrogryposis multiplex congenita appears to be more frequent than generally assumed and, as mentioned before, has been described in several publications. There appears to be agreement on definition of arthrogryposis multiplex congenita as a syndrome characterized by congenital fixa-



Fig. 5. Microscopic: Urinary bladder showing tremendous neurolemmal proliferation partly replacing musculature of bladder wall.

tion of multiple joints associated with a varying number of other congenital anomalies, chiefly of the neuro-muscular and skeletal system. The rigidity of the joints (flexion or extension from birth is pathognomonic of this disorder (Drachman, Banker).[18] However, no general agreement concerning the etiology and pathogenesis has been reached. Some authors (Banker, Victor and Adams)[19] suggest that heredity, virus infection, amyoplasia and primary muscular involvement or intra-uterine pressure must be considered. While no definite familial or genetic pattern is apparent, the condition has been observed in siblings and even in twins both afflicted with the condition (Lipton and Morgenstern).[20] Vester-

mark[21] compares the condition to the Werdnig-Hoffman disease and points out that "Werdnig-Hoffman disease is progressive whereas arthrogryposis multiplex congenita remains stationary after birth." The findings of our own case are suggestive of neurogenic origin as reported previously by those authors who performed autopsy studies, although it should be mentioned that only a few autopsy reports are found in the literature.[6,18,21] The most striking abnormality which has uniformly been described in all autopsy cases is the diminution of anterior horn cells in sections of the spinal cord accompanied by various neuro-muscular lesions. However, to our knowledge the association with neurofibromatosis has not been reported yet. Von Recklinghausen's neurofibromatosis is a well known condition and described in many textbooks; the most comprehensive description is given by A. Schmincke[22] including various unusual features with respect to organ involvement and hydrocephalus as found in our case. The cutaneous manifestations such as cafe au lait spots and neurofibromata, found at birth and slowly progressing, tend to classify this as a heredo-familial disorder (Canale et al.[23]).

Our own autopsy findings as to arthrogryposis multiplex congenita are in essential agreement with previous reports. We were particularly impressed with the obvious reduction in the number of intact anterior horn cells of the cerevical spinal cord without evidence of inflammation. Furthermore, particularly remarkable were myofibrillar atrophy or hypoplasia and irregular increase of fat tissue found in the extremities (sausage-shaped leg). Changes of the peripheral nervous system and hydrocephalus have to be ascribed to Recklinghausen's neurofibromatosis, and in our case a considerable variety of the histologic picture is present ranging between neuroma, neurofibroma and "schwannosis", as defined by Schmincke. The association of myofibrillar atrophy and neurolemmal hyperplasia, as shown in sections of our patient's right calf, may suggest an interrelationship or even common etiology of both conditions and not just mere coincidence. From a pathological viewpoint, however, it should be mentioned that there is a considerable contrast between the essentially atrophic or hypoplastic changes of arthrogryposis multiplex congenita and the proliferative features characterizing Von Recklinghausen's neurofibromatosis which at least makes it difficult to consider a common etiology.

The recently increased attention given to crippled and retarded children may perhaps soon reveal more cases as described above and will enable specialists of all medical fields to continue their studies on this subject.

Summary

A case of arthrogryposis multiplex congenita associated with Von Recklinhausen's neurofibromatosis is reported. The diagnosis was confirmed by clinical and autopsy findings. The chromosome count was forty six and no abnormality was seen in either the number or morphology. From the pathological viewpoint the contrast between the atrophic or hypoplastic changes of spinal cord and skeletal musculature in athrogryposis multiplex congenita and proliferative features in neurofibromatosis are particularly remarkable. No conclusions could be reached as to possible common etiology and pathogenesis.

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American Women in the Prime of Life

The number of adult women in the United States continues to increase more rapidly than the number of adult men. There were over 32.8 million women aged 35-64 in July 1966—a gain of 2.1 million since April 1960 and 6.6 million since April 1950. Their number, moreover, is expected to exceed 34 million in 1975 and to reach 35.5 million by 1980. By contrast, the number of men aged 35-64 may increase from 31 million in July 1966 to 33 million by 1980.

The future growth of the female segment of our population is expected to vary considerably by age group. Most marked is the anticipated rise for women aged 55-64, their number increasing by 25 percent from 9.0 million in 1966 to 11.3 million in 1980. Women at ages 35-39 will also be substantially more numerous in 1980 than at present. On the other hand, the number of women at ages 40-44 is expected to decrease by 9 percent, reflecting the low level of births during the late 1930's.

At the prime of life women will increase faster than men because of their lower mortality throughout the life cycle. This is particularly evident at ages 46-64 where women outnumbered men by about 1,400,000 in 1966; the disparity is expected to widen to over 2,200,000 by 1980. At that time there may be 1,109 women for every 1,000 men at ages 45-64 compared with 1,073 per 1,000 in 1966. (From the Statistical Bulletin of the Metropolitan Life Insurance Company)

Brain Tumors in Psychiatric Practice

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Psychiatric symptoms may be the only ones revealing brain tumors. They are definitely the earliest symptoms. Four patients with brain tumors, diagnosed on psychiatric symptomatology, are presented.

OCCASIONALLY brain tumors are encountered in the psychiatrist's office. It is also found that 2% of all autopsies in mental institutions reveal brain tumors. Psychiatric symptoms could be a part of the symptomatology involved in intracranial lesions, especially in the so-called silent areas of the brain, mainly the frontal lobes. They may even be the only symptoms produced by such lesions. In any way, psychiatric symptoms are the earliest symptoms presented by such patients. Hence, it is extremely important for the psychiatrist to be alerted to early manifestations of these destructive lesions. It is the writer's opinion that intracranial tumors should be diagnosed on psychiatric grounds alone, even before the appearance of neurological pathology. The four cases to be presented in this paper illustrate such a point.

Case #1. Mrs. W. was a white, 65 year old married woman referred by her family physician to the psychiatrist for "putting on forgetfulness". The family physician stated that this woman had become a personality problem since she was not getting along with her husband, forgetting what she was told by him, and at times she forgot some commitments she had made. This led to some marital problems. The attending physician said he had known this patient for many years and quite often he had seen her for what he called "neurotic symptoms". Physical examinations were frequently negative. The patient resented this referral to me. She was quite angry at her husband for bringing her to the office and in the parking lot she was very upset and became nauseated and vomited.

She entered the office, her gait was normal, sat down and asked what she was going to talk about. When she was asked to present her problems as she saw them, she stated that she did not know what her problems were but she surely "didn't feel well". She stated in general that her body was not functioning as it used to. She felt weak and forgot most trivial things at times—most important things at other times. She could not determine exactly the duration of these symptoms. However, she stated she was becoming "increasingly stupider". She concurred that there was a definite change in her personality—approximately within the past six months.

Her conversation was quite relevant and coherent. She was alert at the beginning and related rather well to the examiner in spite of her doubts as to the help she was going to receive. She denied the presence of hallucinations. There was no gross thought disorder. She was well oriented to time, place and person. Her memory was somewhat impaired to recent events, but not to remote events. There was no bizarreness or mannerisms. She was fairly well

From the Mental Health Center of Norfolk and Chesapeake.

dressed. She seemed to present somewhat shallow affect but not inappropriate.

After approximately thirty-five minutes, during the interview, she began to fall asleep. She woke up at the request of the examiner, apologized for having fallen asleep and mentioned that she could not help it, then carried on the conversation. This indicated a disturbance of her sensorium, meaning the level of alertness.

On the basis of the rather sudden increase of her previous adjustment problems and the definite personality changes, the impairment of recent memory and most importantly her sensorium fluctuations, an impression of frontal lobe tumor was made. This was also substantiated by her confusion at times and also the vomiting in the parking lot which was attributed to her being upset. The family physician re-examined her and the findings were entirely normal.

While arranging to send the patient to a University Center where she would receive complete neurosurgical examination, and approximately three weeks later, she developed massive bilateral bronchopneumonia and died within forty-eight hours. Autopsy revealed an ependymoma, grade III malignancy in the left frontal area.

Case #2. Mrs. R. was also 65 years old, a colored female divorced for the past eighteen years from an alcoholic husband who was very much a nuisance and trouble maker. She had been living within the past two years with her daughter and son-in-law. She had had a considerable amount of emotional problems with her two sons. One of them did not work regularly and had problems adjusting to society. The other apparently was an excessive drinker. During the time she was residing with her daughter, a school teacher, and her son-in-law who was under psychiatric care for a schizophrenic illness, she had had a considerable amount of resentment against her daughter who she felt was bossing her. She did not like the attitude of her son-in-law who kept criticizing her. She felt that the couple were belittling and humiliating her. The main complaint of the family was her inability to walk due to emotional problems. She had had this episodic inability to walk within the past year. She was examined at a University Center with no neurological findings. At that time she was released with the recommendation for psychiatric treatment. Her inability to walk became more persistent. The family stated that she could do this at will in order to get attention from them.

Mental status examination revealed no organic manifestations. She was admitted to the hospital to the psychiatric floor. Observation of her behavior revealed that she resented her daughter's treating her an invalid, yet she felt that she could not really walk. She had a fear of death. She stated that some friend of hers with a similar problem had had a spinal tap, an operation, and she died. Hence, she refused any spinal taps or any surgical operation before it was even considered. Neurological examination was not significant. However, the neurosurgeon recommended a pneumo-encephalogram. Skull films were normal, EEG was normal, and brain scan was normal. During that time, the patient felt she was detained in the hospital because they wanted to get rid of her so she made an effort to start walking again. She began, with the assistance of the physiotherapist and the occupational therapist, to walk using a walker. Conditioning rewarding technique was applied.

Finally, she accepted pneumo-encephalogram. There was a dramatic change in her attitude towards surgery. She mentioned that she would rather die than live invalid and requested the neurosurgeon to come see her again.

During injection of the air into the spinal space, the patient had a convulsion. The C.S.F. appearance was normal; pressure could not be measured due to the convulsion. However, the neurosurgeon felt it was high. There was increased intracranial tension. Films taken after the pneumo-enceph-

alogram showed that there was an enlargement in the pituitary region. The neurosurgeon recommended craniotomy for exploration. This was carried out. The pituitary gland was enlarged, there was a tumor mass in the area about the size of an egg—pinkish red in appearance. The tumor mass was pressuring against the third ventricle. The tumor was removed and the patient ran an uneventful course post-operatively. However, she died with a sudden shock on the third day after the operation. The cause of death was undetermined since autopsy was not permitted.

In this case there were numerous psychiatric manifestations. However, they did not seem to be related to her inability to walk. This fluctuating paraplegia could be very easily considered hysterical. However, at this age hysteria should be a less likely possibility than an intracranial lesion. Yet the nature of the course of the paraplegia (fluctuating) was not the ordinary course due to a tumor that would cause paraplegia. These fluctuations could only be explained on the basis of fluctuating intracranial C.S.F. pressure; also, fluctuating pressure within the brain ventricles causing internal hydrocephalus.

Case #3 was a forty-six year old colored woman referred by a former psychiatric patient for "troubles with her nerves". The patient was unable to carry out her daily chores as a domestic. She became more apathetic and unable to sleep and complained of headaches generalized and unrelated to posture. Mental examination revealed intellectual dullness which was commensurate with her education and social function. Her affect was slightly flattened. She was somewhat confused and disoriented. She complained of headaches all over her head; that her nerves were "shot". There were no family problems and no significant emotional stresses.

She was admitted to the psychiatric floor. She was observed as mute at times, bizarre at other times, had episodes of panic, and was paranoid towards the nursing staff and other patients. At one time the nurse reported that the patient would go to sleep at the table while eating with food in her mouth. This was a definite sensorial disturbance. X-ray films were normal, EEG revealed possible right frontal process, particularly a tumor. It read as follows: "Fairly good 8-10 second dominant rhythm of medium voltage, considerably high voltage 2-4 second activity mainly right sided in mono-polar tracings. Bi-polar tracings show maximal slowing bi-frontally and in the right occipital area. No seizure discharges. More diffused slowing with hyper-ventilation. Very slow activity consistent with organic brain disease." This was repeated and produced the same results. Brain scan following the administration of radioactive mercury showed a localized increased uptake in the left frontal lobe indicating probably brain tumor in the left frontal lobe.

This was somewhat confusing since the EEG showed pathology in the right side and the brain scan showed pathology in the left side of the brain. However, cerebral angiogram demonstrated a mass lesion in the left frontal region of the brain.

At the time of surgery a cystic glioma with recent hemorrhage was identified in the mid-portion of the left frontal lobe. Subtotal removal of the tumor with evacuation of the cyst was accomplished. The pathology report revealed glioma of moderate malignancy, probably a glioblastoma multiformae. In this particular patient the mental status examination was highly suspicious of an organic lesion and the laboratory investigations quite readily confirmed such suspicions.

Case #4. Mrs. M. S. was referred to the Mental Health Center of Norfolk and Chesapeake for therapy after being examined by a psychiatrist and a neurologist and was thought to be suffering from "involutional psychosis". She came to the interviewing office in a wheeled chair pushed by her hus-

band. She was wearing dark glasses and complained that the light hurt her eyes so badly. She also had had headaches more on the right side. She stated that light and noise hurt her to the extent she "could die". She looked somewhat odd with shallow affect, somewhat apathetic, slow in answering questions. She also reported a definite personality change—that she had become more retiring and withdrawn than before. Her memory, she complained, was getting worse, she was not well oriented to time, she was confused about the day and the month. She thought it was June at the time of the examination which was August. She was also unable to state her age.

On the basis of these psychiatric symptoms alone a diagnosis of chronic brain syndrome, possibly due to intracranial space occupying lesion, was made. She was admitted to the Norfolk General Hospital and her behavior on the ward was reported as withdrawn, demanding, odd, at times confused and apathetic, yet she was a very nice and likeable person.

She is 57 years old, white—this is her second marriage, no children. She raised her husband's step-daughter and is rather fond of her. There is evidence of neurotic symptoms in the past. The changes in her personality were not an exaggeration of the previous neurotic symptoms, but different. She became withdrawn, retired, refused to take part in family affairs and felt weak and unable to work. Neurological studies, including x-rays, brain scan, right carotid angiogram revealed a tumor mass in the right temporal region. Operation was performed and a tumor about 10x8 centimeters in diameter was removed, a huge meningioma of the right sphenoidal ridge was described by the pathologist. Gradual and complete recovery was the outcome of this patient. She was placed on Dilantin postoperatively by the neurosurgeon. She continues to receive supportive psychotherapy as she finds it difficult to readjust to the changes of her life situation.

Summary

Four patients with brain tumors are described. The diagnoses were made purely on psychiatric symptomatology. These psychiatric manifestations are not too hard to depict once the examiner is oriented towards discovering them. It is my opinion that these are the earliest manifestations of the space occupying lesions of the brain. They usually involve personality changes, exaggeration of previous neurotic symptoms, changes at the level of alertness, unusual symptoms which may not be commonly seen at the particular age or life situation of the patient, early memory impairment, slight disorientation and confusion. Laboratory investigations, x-rays, electro-encephalograms, brain scans and pneumo-encephalograms help confirm or rule out diagnosis. These previous tests should be resorted to in case of doubt or whenever there is a probability of a space occupying lesion. One cannot over emphasize the importance of early diagnosis and treatment of brain tumors.

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Clinicopathological Conference . . . .

Respiratory Failure after Multiple Injuries

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Clinical History

This seventeen year old white primigravida with an estimated date of confinement of November 5, 1967, was in an auto accident on the evening of October 29, 1967. On arrival at the Emergency Room she complained of pain in the right thigh and face, and numbness on the right side of the face.

On physical examination she appeared to be in shock but she was responsive. Blood pressure was 72/40 and the pulse was 110 per minute. There was a large oblique laceration from the left ear to the corner of the mouth with loss of cranial nerve III function on the left side of the face. There was another long laceration extending from the right frontoparietal area to the lateral canthus of the right eye. The neck was supple and the lungs clear. The heart was not enlarged, and no murmur or gallop was described. The abdomen revealed a full term pregnancy with good fetal heart tones. There was swelling and tenderness of the right thigh, with numerous abrasions over the legs. Pulses were normal in both legs.

Laboratory values on admission revealed a hematocrit of 34.5%, and a white blood count of 23,250. Urinalysis showed a specific gravity of 1.028, and there was no protein, sugar, or significant cellular debris. On October 30, 1967, platelet count was 201,000 per cubic millimeter, fibrinogen level was 65 mgm. per cent, euglobulin lysis time was less than 12 minutes, a factor VII assay was 100%, partial thromboplastin time greater than three minutes, and prothrombin time was 15.5 seconds/11 seconds. On the afternoon of October 30, 1967, fibrinogen level was 225 mgm. %, and euglobulin lysis time was 25 minutes.

Whole blood transfusions were begun immediately, and she was taken to the Operating Room where repair of the buccal branch of the left facial nerve and closure of the facial lacerations were carried out. A Kirschner wire was inserted through the proximal tibia. Near the end of the procedure, severe respiratory distress developed, copious quantities of thin secretions appeared in the tracheobronchial tree, shock increased with fall in blood pressure to 60 millimeters of mercury systolic, and fetal heart tones were lost. Later it was determined that the protein content of the tracheal secretions was 1.9 grams per cent. She subsequently required vasopressors, occasional plasma, and whole blood. On October 31, 1967, her platelet count was 73,000 per cubic millimeter and on November 4, 1967, was 9,000 per cubic millimeter. Fibrinogen levels and euglobulin lysis time remained within the normal range after the initial values, but prothrombin time was about five seconds elevated. Electrolytes were maintained within the normal range.

Multiple blood gas determinations done before and after the patient was tracheotomized and on the Engstrom respirator showed consistently low pO_2 (20-50 millimeters of mercury) and usually normal pCO_2 (35-45 millimeters of mercury), in spite of many adjustments in ventilator flow rates and pressures, and in O_2 concentrations in the inspired air. On October 30, 1967, plasma volume was 1680 cc., total blood volume was 2516 cc., and red cell mass was 1258 cc. On October 30, 1967,

electrocardiogram showed a QRS axis of +130 degrees, and non-specific ST-T wave changes. On November 1, 1967, the QRS axis had shifted to +100 degrees with no other changes.

In spite of hypothermia, digitalis, and antibiotics, she did not improve significantly. Prednisone was given intravenously. Tracheal instillations of acetylcysteine, 50% ethyl alcohol, and saline were used. Adequate ventilation was never possible, and she died in shock on November 5, 1967.

*Dr. Douglas Eastwood:** A seventeen year old girl, pregnant and almost at term, was admitted to the Emergency Room following an auto accident, complaining of pain in the right thigh and face, and numbness of the right side of the face. She appeared to be in shock but was responsive.

This statement "appeared to be in shock" should be considered carefully. What caused this girl to have a "shocklike" appearance? She was undoubtedly pale with cold skin; she may have been apprehensive, a feeling intensified by the action of circulating catecholamines secreted to compensate for the loss of circulating blood volume by producing vasoconstriction. She may have exhibited air hunger, tachypnea, and hyperventilation. The stimulus for this respiratory response is cerebral hypoxia and a significant metabolic acidosis as a result of inadequate perfusion of vaso-constricted areas of the body. An arterial pressure measurement of 70/40 confirms the drop in perfusion pressure. A pulse of 110/minute is elevated but not extremely rapid.

It should be remembered that face lacerations of the extent here described are usually associated with profuse blood loss. Examination of the abdomen showed it to be distended with the full-term pregnancy, and good fetal heart tones were detected. The baby was alive and apparently not in distress at this point. Now, it has been demonstrated that uterine blood flow is certainly dependent on arterial pressure. Fetal distress develops during hypotension produced by high spinal anesthesia. Drugs such as norepinephrine or neosynephrine given in the treatment of spinal hypotension will increase rather than correct fetal distress. In addition to this maternal control of the circulation, the placental-fetal unit apparently also has some control of uterine blood flow. A hormone secreted by the placenta to which uterine vessels are very responsive appears to be capable of producing significant uterine vasodilation despite the maternal neurogenic or hormone stimulus. In the patient under discussion, we have no evidence that the placental-fetal uterine unit was disturbed prior to surgery. There may have been a period of vasoconstriction with inadequate perfusion, but, at the time of admission, the fetus appeared to be viable.

Further examination revealed swelling and tenderness of the right thigh. The presence of abrasions, bruising, and swelling is of clinical significance in attempting to delineate the cause for the patient's subsequent clinical picture, because it indicates tissue trauma, blood loss, and blood vessel damage in several areas of the body.

Before consideration of the laboratory picture and x-rays, I believe we should examine retrospectively the decision which had to be made by the surgeon. It was necessary for him to balance the result in a young attractive girl of an inadequate repair of large scar and a severed facial nerve under local anesthesia against the risk of the necessarily-long immediate surgical repair. Included in this risk are a general anesthetic in an emergency situation, prolonged fixation of the patient in one position, the inability to follow symptoms as well as signs to evaluate the patient's changing status, and the additional transfusions necessary to replace the surgical blood loss. The anesthesiologist is faced with a pregnant patient who has been, and probably still is, in shock and in whom the replacement of intravascular and extra-

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cellular fluid has not been complete. The surgery must be performed on both the face and the leg, and the patient must be considered as having a stomach full of fluid and swallowed blood.

It was elected to proceed with the operation; whole blood transfusions were given, the facial lacerations were repaired, and the facial nerve was approximated. A Kirschner wire was inserted through the proximal tibia for traction. About two and a half hours after the start of surgery, and during the administration of the fifth unit of blood, the surgeon noticed the sudden onset of capillary bleeding, necessitating his reopening an already closed wound in an attempt to staunch the flow. No single large vessel bleeder was found but cautery and ligation were successful, permitting a reclosure.

An hour and a half later, there was the sudden onset of copious, thin secretions from the endotracheal tube. The patient's systolic blood pressure fell to 60 millimeters of mercury and the fetal heart tones, which had been monitored through a stethoscope taped to the abdominal wall, disappeared. Without looking at the information subsequently obtained, we should consider the possible diagnoses facing the anesthesiologist as to the cause for these secretions. The first consideration was *left ventricular failure* on the basis of previously undiagnosed heart disease, direct myocardial trauma, or, possibly, overload of fluid volume. *Chemical pneumonitis* from aspiration of acid gastric contents was considered. *Pulmonary-capillary* damage with transudation of fluid without left heart failure was a possibility. The fluid volume which had been administered up to this time included 4,500 cc. of whole blood and 6,000 cc. of Ringer's lactate solution. The central venous pressure which had not previously been monitored was found at this point to be 150 mm. of water. Ventilation of the patient required high airway pressures and prolonged inspiration time, indicating poor pulmonary compliance and increased resistance. Laboratory data indicate a hematocrit of 34.5% on admission and a variation of between 30 and 43 thereafter. It fell to some degree after surgery and more so after delivery, but *no evidence of overtransfusion was found.*

A white blood count of 23,250 taken shortly after admission to the Emergency Room and prior to surgery is of significance. A white blood count may be high in the absence of infection, in association with the onset of labor, excessive exertion, excitement, or acute hemorrhage. It is probably the acute hemorrhage and excitement in this circumstance which caused this elevation.

Various platelet counts as found in the patient's hospital record include the following: 10-30-67, 7:00 a.m., 200,000—9:30 a.m., 211,000—10-31-67, 9:00 a.m., 73,000 —11-4-67, 9,000. Since the transfusions given during surgery were fairly recently drawn and one bottle was fresh blood, it seems unlikely that low platelet counts as a result of transfusions could have been the sole cause for the hemorrhage at 5:30 a.m., especially since the count was 200,000 at 7:00 a.m. The subsequent fall to 73,000 on the first post-surgical day may be explained by platelet incompatability which commonly follows multiple transfusions and by the consumption of platelets. The subsequent fall to 9,000 may be explained by the fact that the patient's temperature fell to 30 degrees centigrade. At this temperature, the platelet count falls as a result of sequestration rather than destruction, since warming will bring the platelet count rapidly to a normal level.

Apparently a reasonably normal fibrinogen level was present at 5:30 a.m. on 10-30-67 since at that time a clotting time was eight minutes. A good clot developed which retracted and there was no lysis of this clot in five hours. By 7:00 a.m., however, fibrinogen was "immeasurably low". At 9:30 a.m. the value was 65 mgm. per cent, a significantly low level, but by 2:00 p.m. the blood level was 225 mgm. per cent, and the

following day it was at 280 mgm. per cent. Thus, something happened to cause the loss of fibrinogen between 5:30 and 7:00 a.m. A euglobulin lysis time of less than 12 minutes indicated the presence of circulating fibrinolysins at 9:30 a.m., but, by 2:00 p.m. in the afternoon, fibrinolysin activity had fallen to an insignificant level.

Other clotting factors studied include a partial thromboplastin time of greater than three minutes, on 10-30-67 an abnormal measurement indicating that one or a number of the factors necessary for formation of thromboplastin were inadequate. Prothrombin times were slightly elevated as a result of the hemorrhage, and hypoprothrombinemia is a common manifestation of hemorrhage. Factor VII was found to be present at the 100 per cent level. This prothrombin conversion accelerator is a necessary component of the intrinsic coagulation system. Also of significance in the laboratory report is the fact that the total protein was reduced, and it was the albumin fraction that was significantly reduced below normal. Albumin is a smaller molecule, more easily lost from the circulation through damaged capillaries. The plasma-like transudate bubbling from the patient's endotracheal tube to a total volume of approximately 2,000 to 2,500 cc. in twelve hours represents the more likely source for this protein loss.

Multiple blood gas determinations were made which in general indicated a low arterial oxygen tension in spite of very high inspired oxygen tensions. The hyperventilation necessary to raise arterial oxygen tension washed out the carbon dioxide so that it was necessary to add CO_2 to the inhaled atmosphere to maintain a normal arterial carbon dioxide tension. Increasingly high pressures were required on the ventilator to maintain a gas exchange. Blood volume measurements obtained in the postoperative period, because of suspected overhydration, indicated, actually a significant *reduction* below predicted levels in the circulating red cell mass and plasma volume. The textbook, "Fundamentals of Clinical Hematology" by Leavell and Thorup, indicates that in pregnancy there is an 800 to 1300 cc. increase in total blood volume. It is evident that this patient did not have an overloaded circulation at the time the blood volume determinations were made. Urine examination with Sudan IV stain on 10-30-67 showed a small amount of fat. Subsequently, the urine showed innumerable red blood cells which may have been the result of parenchymal renal damage or lower urinary tract trauma at the time of catheterization or delivery. May we see the x-rays?

Dr. William Craddock: Initially, a comminuted fracture of the mid-shaft of the femur is seen; subsequently good alignment is noted. There is no skull fracture, but soft tissue injury is apparent. A fetus is seen in the pelvis. The chest appeared clear. On November 1, 1967, a portable film is quite suggestive of pulmonary edema, although a central interstitial exudate cannot be ruled out. Later, this picture worsens with densities in an alveolar pattern, compatible with almost any form of cardiac or non-cardiac pulmonary edema; however, the azygos vein does not appear dilated.

Dr. J. Edwin Wood, III: The chest films are similar to those seen in high altitude pulmonary edema.

Dr. Douglas Eastwood: Subsequent therapy consisted of continued artificial ventilation of stiff, non-compliant, obstructed lungs. Additional plasma, fluids, and blood were given as needed. Antibiotic therapy was started using penicillin and kanamycin. Prednisone was administered, metaraminol was given to support the blood pressure on occasion, ethacrynic acid was added to stimulate urine formation, and later digitalization was carried out because of decreasing arterial pressure and terminally rising venous pressure. On 10-30-67, consideration was given to the use of a veno-arterial pump-oxygenator for a period of time to permit

adequate tissue oxygenation. It was elected to use hypothermia and sedation to reduce the oxygen demand since adequate oxygenation could not be provided through the pulmonary tree. The stillborn fetus was delivered on 10-31-67. The placenta did not appear to be abnormal.

The patient died on 11-5-67. Her death was caused by the event that occurred sometime shortly after 5:30 a.m. on 10-30-67. Considerations as to possible causes of this event can be classified as follows:

A. Pulmonary edema on the basis of cardiac failure. Against this diagnosis are the fact that the patient showed no previous signs of cardiac disease. In the presence of a normal myocardium, anesthetic drugs are unlikely to produce significant myocardial depression. There was no evidence of direct trauma to the heart. An acute overload should be considered, particularly since there is a possibility of a high blood volume in pregnancy. There may also be a small pulmonary volume because of the elevation of the diaphragm, particularly with the patient in the supine position. At one time in the postoperative period, the endotracheal tube was found to extend into the right main stem bronchus obstructing the left main stem bronchus. If this had occurred during surgery, inadequate ventilation of the left lung and associated vasoconstriction of the pulmonary vascular bed on that side could have occurred.

B. Direct lung injury at the time of the accident or aspiration of gastric contents with chemical pneumonitis could have produced extreme bronchospasm and secretion formation. These secretions are more blood-tinged and are not as voluminous.

C. Pulmonary-capillary injury as a result of emboli may have occurred. Amniotic fluid emboli have been the cause of death in parturient patients. It is the particulate material rather than the fluid forced into the circulation which produces the problem. Fat embolization must be considered and further discussion of this possibility will follow. Intravascular coagulation, on a disseminated or even somewhat localized basis, may be the cause for multiple small pulmonary emboli. Multiple transfusions were given, and in all probability with each transfusion there is some degree of incompatability; some degree of intravascular coagulation and some amount of embolization occurs. Of the above possibilities, my formulation is as follows:

1. Pulmonary edema occurred as a result of clot embolization to the pulmonary capillary bed. The acute obstruction of the pulmonary circulation produced *reflex* bronchospasm with resulting resistance to ventilation.

2. The clots which embolized were formed by disseminated intravascular coagulation as the result of: (a) hypercoagulability during pregnancy, (b) the metabolic acidosis of shock, transfused blood, and cold, (c) tissue trauma and vascular trauma in various areas, particularly the leg and face, and (d) transfusion reaction probably not of major proportion.

3. Associated with the coagulation consumption of platelets and fibrinogen, there was also instability of lipid suspended in the circulating blood. There was coalescence of the fat to form globules which embolized. There was also red blood cell rouleaux formation and agglutination. The fibrin, the fat, and the blood cells plugged the capillaries particularly in the pulmonary-capillary bed. These particles probably also lodged in the brain, in the kidney, and other tissues. This patient was in a coma immediately after the procedure and then roused. Kidney function was poor immediately after surgery and then returned. In each of these organs there was the capacity for restoring normal function. The lung, however, was unable to recover. Because of the inadequate pulmonary perfusion due to the emboli, a hypoperfusion syndrome similar to that seen in hyaline membrane disease of the newborn developed.

The pathological examination, therefore,

will show, I believe, atelectatic lungs which are consolidated and congested. The uterus will show a normal postpartem endometrium. The kidney perhaps will show old emboli but without marked damage. The brain, heart, liver, etc., will not be remarkable except for small emboli. Emboli composition will be largely fibrin clot though there may also be fat globules and red blood cell agglutination.

Dr. Douglas Eastwood's Diagnosis:

1. *Wide-spread intravascular clotting, secondary to trauma and hypoxia.*
2. *Multiple emboli to lung, brain, and heart.*

Dr. David E. Smith: The lesions relative to the interpretation of this case are of several types and scattered sites. To keep their presentation from being confusing, let me begin by stating my conclusion that they support the interpretation Dr. Eastwood has just given. This patient suffered essentially from capillary and endothelial damage that was provoked by shock and respiratory insufficiency. There was both gross and microscopic intravascular thrombosis and, additionally, intravascular conglomeration of neutral fat, generally referred to as fat embolism. The course was complicated by at least one finding revealed only by the autopsy: a wide-spread intrapulmonary growth of *Candida albicans.*

The lungs were uniformly and diffusely heavy, rubbery, firm, and dark purplish-red. A few small branches of the pulmonary arteries contained gross thrombi. The most striking change was apparent microscopically where thick acidophilic membranes lined the alveoli and particularly the bronchioles and alveolar ducts. (Fig. 1) The membranes were accompanied by erythrocytes, macrophages, pneumonocytes, and small fragments of fibrin in the alveolar lumens, as well as there being marked congestion of the alveolar capillaries and moderate infiltration of lymphocytes and macrophages into the alveolar septa. The total picture was that of a diffuse interstitial pneumonia with marked hyaline membrane formation. There was no evidence of amnionic debris in any blood vessels.

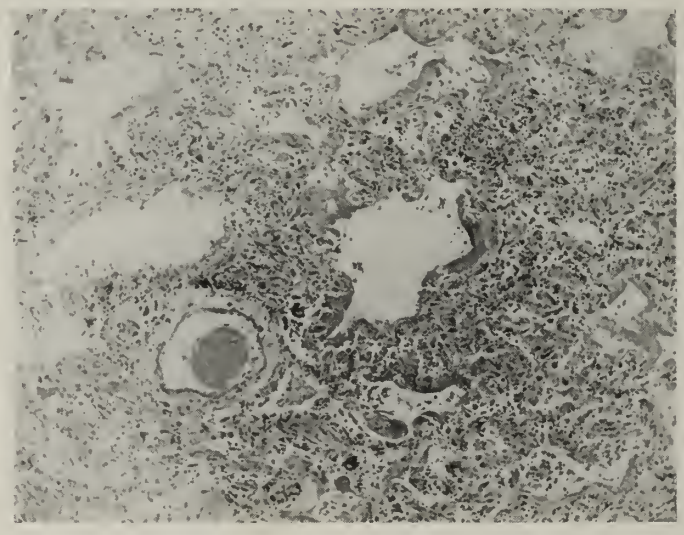


Fig. 1. Hyalin membranes in the alveoli, congested capillaries, and interstitial inflammation in the lung. A non-occlusive fibrin thrombus occupies a small vessel in the lower left part of the illustration. (H. & E. stain, 60X.)

Although the fundamental pathogenic process in this case, intravascular clotting and consumptive coagulopathy, was similar to that in cases of amnionic fluid embolism, there was no evidence that amnionic fluid

played a part in the onset of the syndrome.

In numerous parts of the lungs, particularly where alveolar walls seemed to have undergone bland necrosis, there was a widespread growth of *Candida albicans* within the alveolar walls, fibrinous masses, and membranes. (Fig. 2) The areas of necrosis were probably related to vascular obstructions which were of several types. In addition to thrombi in small and large vessels (Fig. 1), there were many masses of fat stainable by Sudan IV in frozen sections. Similar droplets of neutral fat were easily demonstrated in the kidney and in the brain. (Fig. 3) These findings are customarily referred to as fat embolism. As Dr. Eastwood has pointed out, the mechanism is more probably a conglomeration of chylomicrons under the influences of the disturbances that are precipitating intravascular



Fig. 2. Yeast forms of *Candidia albicans* growing in the hyalin membranes, necrotic septa, and alveoli of an area of necrosis in the lung. The fungi are the numerous, dark, hollow, round bodies less than 1 mm. in diameter in this illustration. (H. & E. stain; 260X.)



Fig. 3. "Fat embolus" in the brain. Just above and to the right of the center a dark cylindrical mass that stains red with Sudan IV fills a segment of a capillary. (Frozen section, Sudan IV stain, X160.)

clotting than a true embolization from a site of injury.

From thrombi in the pelvis about the postpartum uterus, and possibly from other sites such as the lower extremities, there were emboli that had lodged in small pulmonary arteries. More important, however, were the thrombi in very small vessels throughout the lungs. These were composed principally of fibrin and often not occlusive. They were also detected in small vessels in the submucosa of the colon. Because of the limits of examination by the light microscope, we probably overlooked further deposition of fibrin in thin layers along the endothelial lining of vessels throughout the body. The small thrombi we did demonstrate are acceptable evidence that intravascular coagulation occurred.

Another evidence of intravascular coagulation consisted of large, friable, fibrinous vegetations along the line of closure of the tricuspic valve: a grossly typical lesion of thrombotic nonbacterial endocarditis. There was also a thin gross layer of fibrinous clot that covered a large part of the endocardium of the right antrum and extended into the vena cava. This was probably a quite terminal event but might be interpreted as evidence of a continuing intravascular clotting.

The heart contained two additional findings. Beneath the endocardium of the left ventricle there were rather wide-spread ecchymoses. The extravasation of blood at this site of constant motion is commonly interpreted as due to rupture of capillaries that have been damaged by hypoxia. In a case such as this, where we know how severely hypoxic the patient was, it was easy to accept this explanation. Additionally, throughout the myocardium there were many examples of leukocytes adherent to capillary walls and other leukocytes that had actually migrated into an interstitial position. This appeared to be another manifestation of the diffuse vascular damage.

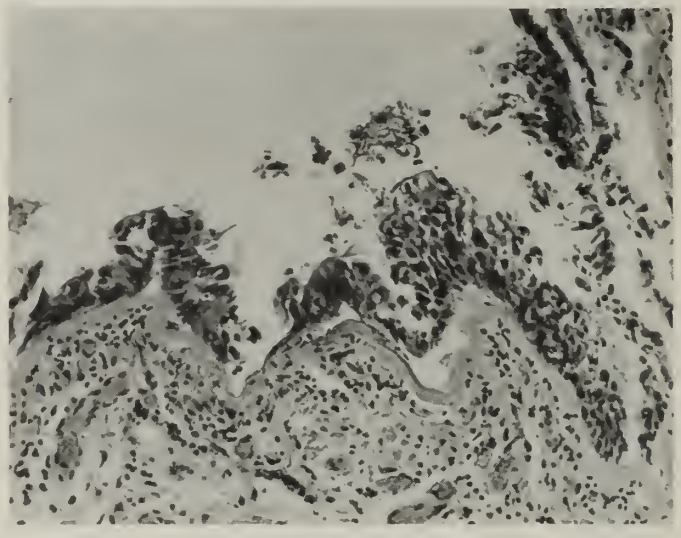


Fig. 4. Squamous metaplasia of epithelium in a small bronchus. The irregular, dark nuclei and cells can be compared to the lesion that has been called carcinoma-in-situ of the bronchus, yet in this case they are obviously the result of the inflammation of the lungs in this young woman. (H. & E. stain, X160.)

To return to the lung, I would like to point out that there was a rather marked metaplastic reaction in the linings of the small bronchi. (Fig. 4) The transformation of columnar epithelium to a squamous type, with considerable irregularity of cells, undoubtedly occurred because of the widespread inflammation in these lungs, although the picture might tempt some to compare the appearance to that of carcinoma-in-situ. Any suggestion of that diag-

nosis would appear entirely without foundation in this seventeen year old girl who had no indication of antecedent disease.

The evidence at autopsy, then, indicated a marked disturbance of plasma clotting factors and the proper function of capillary walls. This resulted in intravascular fibrin thrombi and so-called fat emboli. In addition, the alveoli of the lung were lined by dramatic and thick hyaline membranes. These were of the type one sees in prolonged cases of mechanical respiration, particularly when high oxygen concentrations are administered. In this case, the historical events would suggest that the lesion had begun with the initial dramatic episode of pulmonary transudation during the shock at the end of the operation on the first night. The process was undoubtedly complicated by the later hypoxia and oxygen administration. The vitality of the lung was so damaged by this process that it allowed the wide-spread growth of *Candida albicans,* a fungus that is usually a saprophytic surface contaminant.

Dr. Thomas H. Hunter: Do you mean that the lesions you demonstrated, that we have always called fat emboli, are not to be thought of as true emboli, despite the fact that there was a fracture of a long bone?

Dr. David E. Smith: Yes, they seem to be more a product of an altered physical state of the plasma fat, ordinarily suspended as chylomicrons, rather than physical masses arising at the site of the injury. A number of relations between plasma fat and coagulation factors are well known, if not well understood. This interpretation is particularly attractive in explaining the presence of the masses in capillaries of the brain and kidney beyond the pulmonary bed.

Dr. Douglas Eastwood: They are really just part of the total process. These injured patients have shock, respiratory insufficiency, hypoxia, endothelial damage, acidosis, and develop intravascular coagulation and disturbance of the physical state of colloids in their blood stream. I do not believe that a separate syndrome of fat embolism exists without the other elements of this picture.

Dr. Munsey Wheby: It is clear now that this patient should have been heparinized.

Anatomical Diagnoses:

Multiple traumatic injuries, including femoral fracture;
Fibrin thrombi in the lungs and gastrointestinal tract (intravascular coagulation syndrome);
Diffuse interstitial pneumonia with hyaline membranes;
Bronchopneumonia with Candida albicans;
Thrombotic nonbacterial endocarditis on the tricuspic valve.

Cancer Trends . . . .

Edited by—
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Early Diagnosis of Lung Cancer

GEORGE R. MINOR, M.D.

The rapid increase in incidence of cancer of the lung during the past 50 years is generally agreed to be related to chronic irritation of the bronchial mucosa. It seems self-evident that cigarette smoke is the most important of the inhalant irritants, quantitatively if not qualitatively. If we cannot radically alter the smoking habits of adults, we do have the responsibility of utilizing to the fullest the methods of early diagnosis of lung cancer which are available to us.

Of recent diagnostic aids, cytologic study of the sputum has seemed quite promising. Unfortunately, the logistics of producing and delivering appropriate specimens to the cytologist are not simple, nor are the results always reliable. False positives as well as false negatives are not uncommon. Positives (class IV, V), if they are to be regarded as significant, should be repeatedly confirmed. Negatives have little significance and certainly should not influence the clinical decision as to confirmation of the diagnosis and treatment.

Mediastinoscopy has been proposed as an innocuous extension of prescalene exploration. The finding of positive mediastinal nodes is regarded by some as a contraindication to thoracotomy. Certainly it is justifiable to eliminate thoracotomy as a purely diagnostic procedure, but too often this becomes the basis of a negative approach. It is well established that cancer not only can be cured by surgical excision, but that over 25% of the five-year survivors following resection have had positive hilar or mediastinal nodes at the time of surgery. The simple presence, then, of a positive node should not alone necessarily contraindicate a thoracotomy.

Needle biopsy has been done for a number of years. With the help of fluoroscopic control, a histologic diagnosis can be obtained on most lesions, large or small, peripheral or central, with a minimum of morbidity and a fair degree of accuracy. It is questionable, of course, whether a positive preoperative diagnosis in a resectable lesion is necessary, though it can obviously save valuable operative time. As in the cytologic study of sputum, the negative may be a false negative and should not influence the decision to explore. The greatest usefulness of needle biopsy is probably the histologic proof of metastatic disease and its possible origin, or the identification of the inflammatory lesions which need not be resected if they can be differentiated from cancer.

Special radiographic studies such as laminography may reveal central or concentric calcium in small nodules, which is sufficient proof of their granulomatous nature to eliminate exploration and excision-biopsy. Many times, however, laminography does not establish the presence of calcium characteristically located. In this case *the lesion should be removed*, since, regardless of what the radiologist or surgeon may wish to think, it will not infrequently prove to be carcinoma, especially in patients over 30. A review of all previous films, if any exist and can possibly be obtained, is probably as valuable as laminography.

Conventional posteroanterior roentgenograms of the chest remain our most valuable diagnostic tool. Despite the fact that cure

MINOR, GEORGE R., M.D., *Professor of Surgery, Department of Surgery, University of Virginia Medical Center, Charlottesville.*

depends on many factors as yet poorly understood, such as degree of malignancy and resistance of the host, there can be no doubt that the earlier a lesion can be found and removed, the greater the possibility of cure. Both cure rate and resectability rate are greatly enhanced if the lesion is a small asymptomatic nodule. The contrast between a solid nodule and the surrounding radiolucent lung permits the lesion to be seen when it is less than 1 cm. in diameter. Under these circumstances it is surprising that more physicians do not make use of the chest roentgenogram as a routine extension of the physical examination at regular intervals. It is a more productive survey tool than the hemogram or urinalysis, which have been minimum standards of satisfactory practice for over 50 years. It seems only proper, therefore, that patients over 40 have an annual chest film as part of their physical examination, and that hospitals require admission films on everyone over 18 unless the patient has had a negative film at the same institution within the past six months. An important by-product is the discovery of other pulmonary and cardiovascular lesions.

Comments

We are constantly seeking more effective means of detecting cancer of all sites at an earlier stage of development. Lung cancer is rapidly increasing in incidence and the overall results of treatment are disappointing. Nevertheless, detection of lung cancer at a stage that is amenable to resection leads to a significant number of long survivals. Dr. Minor's remarks are most timely, as he reminds us that, despite more recent procedures, our best diagnostic tool is still the periodic chest x-ray. We can only re-emphasize this point and urge physicians to utilize this approach to the asymptomatic patient at regular intervals. Although this message is not a departure from current thought, it is worth stressing in view of justifiable pessimism stemming from the currently high incidence of non-resectable lung cancers.

THE EDITORS

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Let's Reminisce!

SEATS FOR SHOP GIRLS—*The Independent Practitioner* is our authority—and high authority too—for stating that New York now requires "shop-keepers to prepare seats for females in their business places." *The Practitioner* properly adds, "The enlightened intelligence of the age is rapidly pointing out, or indicating, many new or heretofore unutilized methods and ways of preserving health." If we may add that if our Legislatures and Councils would heed the advice of an enlightened profession, then many of the ills of humanity would be abated. But we note "progress". (Virginia Medical Monthly, August, 1881)

Diagnostic Laboratory Medicine . . . .

M. R. ESCOBAR, Ph.D.
M. J. ALLISON, Ph.D.
H. P. DALTON, Ph.D.

A_2-Influenza Outbreak at the Medical College of Virginia

In anticipation of the predicted outbreak of Asian (A_2) influenza for the 1967-68 season, the Virginia Department of Public Health requested the assistance and participation of the Virus Diagnostic Laboratory at the Medical College of Virginia (MCV) in the Influenza Surveillance Program throughout the State.

The Student and Employee Health Services of MCV reported 16 cases of influenza-like illness within a two-week period starting on December 28, 1967, and ending on January 12, 1968. Throat wash specimens and paired sera (drawn in the acute and convalescent phases of the disease) were submitted for testing to the MCV Virus Diagnostic Laboratory.

Laboratory diagnosis of influenza type A was first established at the MCV Virus Diagnostic Laboratory on the basis of at least a fourfold rise in antibody titer of paired sera using the Complement-Fixation (CF) test. Confirmation of this diagnosis and further characterization of this outbreak as A_2-influenza were carried out by the Department of Health on the basis of at least a fourfold rise in antibody titer of paired sera using the Hemagglutination-Inhibition (HI) test. Virus isolation was also attempted in rhesus primary monkey kidney cells and embryonated eggs unsuccessfully. It should be noted that isolation of A_2-influenza virus from cases confirmed serologically in other parts of the country has been relatively rare during the recent epidemic season.

TABLE I

SEROLOGICAL FINDINGS IN THE FIRST GROUP OF PATIENTS

PATIENT	Specimen	Collection Date	CF-Antibody Titer*		HI-Antibody Titer*	
			A-antigen	B-antigen	A_2-antigen	B-antigen
1	Acute	1/ 3/68	8	8	40	20
	Convalescent	1/15/68	32	8	640	20
2	Acute	1/ 9/68	8	8	40	20
	Convalescent	1/22/68	128	8	640	20
3	Acute	1/12/68	8	8	20	20
	Convalescent	1/31/68	128	8	80	20
4	Acute	1/10/68	16	8	20	40
	Convalescent	3/11/68	64	8	80	40
5	Acute	1/ 3/68	8	8	10	20
	Convalescent	3/15/68	64	8	40	20

*Complement-Fixation (CF) and Hemagglutination-Inhibition (HI) titers are expressed as the reciprocal of the highest serum dilution giving a positive reaction.

Serological findings are tabulated in three groups according to: (1) fourfold rise in antibody titer of paired sera with both the CF and the HI test, (2) fourfold rise in antibody titer of paired sera with

either the CF or the HI test, and (3) titers of 128 or above for sera collected during the convalescent period in cases when the acute serum had not been made available. Results for influenza, type B, are also included for comparative purposes.

The first group consisting of five patients is shown in Table I. The diagnosis of influenza type A was readily made on these patients on the basis of the CF test alone. At least a fourfold rise in HI antibody titer of their paired sera further helped to specify their illness as A_2-influenza.

The second group consisting of six patients is listed in Table II. Two of these patients had at least a fourfold rise in CF antibody titer of their paired sera, but only a twofold rise in HI antibody titer. Although a twofold rise is inconclusive in determining current or present illness on the basis of one test alone, the identification of these illnesses as A_2-influenza can be safely made in the light of results with both serological tests. Two additional pairs of sera had a fourfold increase in CF antibody titer but in the absence of any HI antibody rise. One could conclude that these patients had A-influenza; however, the diagnosis of A_2-influenza may only be presumptive in nature. Unfortunately, sera on the remaining two patients were made available only for CF testing with eightfold rise in antibody titer. Since the HI test had not been performed on these patients one could only establish the diagnosis as A-influenza.

The third group consists of three patients appearing in Table III. No acute sera were made available in these cases, but both the CF and the HI titers were significantly high to indicate at least a recent infection in two of these patients.

There were two additional patients not included in the table whose sera had no significant rise in titer although clinically diagnosed as having an influenza-like illness.

The CF test antigen used in this study was of the soluble type and did not react with strain-specific antibodies. The CF test using this antigen merely serves to differentiate influenza A from influenza B; antibodies to these types appear early in the course of the illness. However, a CF test antigen other than the soluble type may be used to characterize the A_2-strain of influenza. The antibodies to this antigen appear later during the illness and are identi-

TABLE II

Serological Findings in the Second Group of Patients

Patient	Specimen	Collection Date	CF-Antibody Titer*		HI-Antibody Titer*	
			A-antigen	B-antigen	A_2-antigen	B-antigen
6	Acute	1/10/68	8	8	80	10
	Convalescent	1/24/68	64	8	160	10
7	Acute	1/ 8/68	8	8	40	80
	Convalescent	1/26/68	16	8	80	80
8	Acute	12/29/67	16	16	ND†	ND†
	Convalescent	1/19/68	256	16	ND†	ND†
9	Acute	12/28/67	16	32	ND†	ND†
	Convalescent	1/18/68	256	32	ND†	ND†
10	Acute	1/ 8/68	8	8	40	40
	Convalescent	1/26/68	128	8	40	40
11	Acute	1/ 8/68	8	8	40	10
	Convalescent	1/26/68	128	8	40	10

*Complement-Fixation (CF) and Hemagglutination-Inhibition (HI) titers are expressed as the reciprocal of the highest serum dilution giving a positive reaction.
†ND = Not done because specimen was not available.

fied with the HI antibodies used in this study. One should, therefore, specify the type of antigen used in the test when making statements of whether the CF or HI antibodies appear earlier during the infectious process. In contrast to influenza, HI antibodies appear earlier than the CF antibodies, for example in the case of rubella. These facts may be taken into consideration to strengthen the diagnosis of patients in the second group as A_2-influenza even though only a twofold rise in HI antibody titer was noted.

These findings confirm an outbreak of influenza caused by A_2-influenza virus in the Medical College of Virginia within a two-week period. Only two cases diagnosed as influenza-like illness clinically failed to receive a laboratory confirmation.

TABLE III

Serological Findings in the Third Group of Patients

Patient	Specimen	Collection Date	CF-Antibody Titer*		HI-Antibody Titer*	
			A-antigen	B-antigen	A_2-antigen	B-antigen
12..	Convalescent	1/10/68	128	8	80	10
	Convalescent	1/19/68	128	8	80	10
13..	Convalescent	1/10/68	128	32	80	40
	Convalescent	1/18/68	128	32	80	40
14....	Convalescent	1/ 2/68	128	8	ND†	ND†

*Complement-Fixation (CF) and Hemagglutination-Inhibition (HI) titers are expressed as the reciprocal of the highest serum dilution giving a positive reaction.
†ND = Not done because specimen was not available.

Division of Clinical Pathology
Medical College of Virginia
Richmond, Virginia

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These statistics are but a small percentage of the data contained in "Attitudes and Platitudes" the 1968 edition of The Travelers annual booklet of highway accident information. The statistics are compiled from records of state motor vehicle departments.

The booklet shows that 52,200 persons died on the highways in 1967. More than 4,200,000 persons were injured. More than 40,000 persons were killed on clear, dry roads. The 20 to 34 age group stands the greatest chance of being killed in auto accidents. Speed is still the greatest killer.

There are many ways in which motorists can help try to break the long chain of events that lead to a traffic accident. They include: update driving skills; practice defensive driving; maintain the car in good mechanical condition; limit driving when tired, ill, emotionally upset and under medications; don't drive under the influence of alcohol; adjust speed to highway, weather, traffic and other conditions; and support better law enforcement, highway improvement, improved driver licensing, etc.



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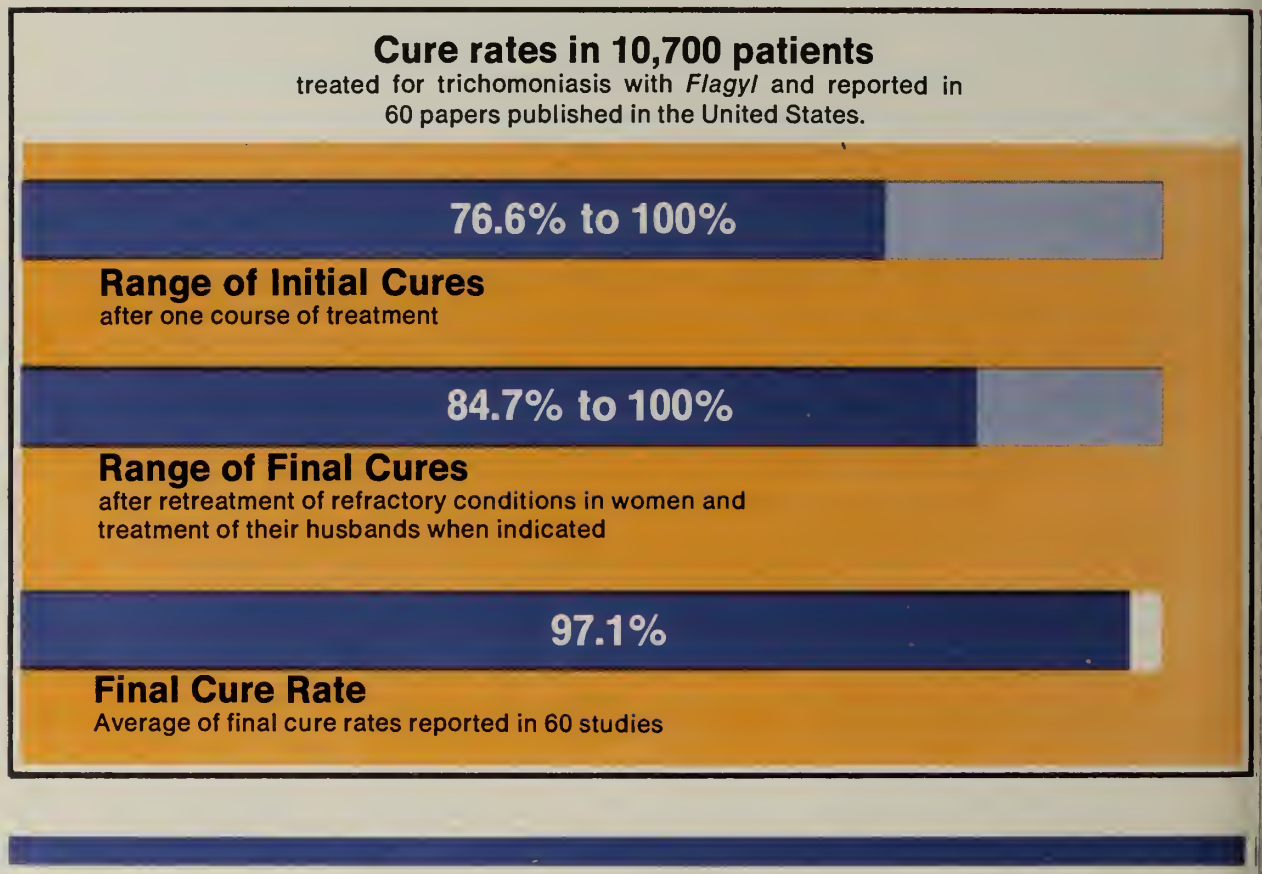
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dominal cramping, constipation, stomatitis, numbness or paresthesia of an extremity, joint pains, confusion, irritability, weakness, cystitis, pelvic pressure, dyspareunia, fever, polyuria, incontinence, decreased libido, nasal congestion, proctitis and pyuria. Elimination of trichomonads may aggravate candidiasis.

Dosage and Administration: In women: one 250-mg. oral tablet three times daily for ten days. A vaginal insert of 500 mg. is available for local therapy when desired. When used, one vaginal insert should be placed high in the vaginal vault each day for ten days; concurrently two oral tablets should be taken daily.

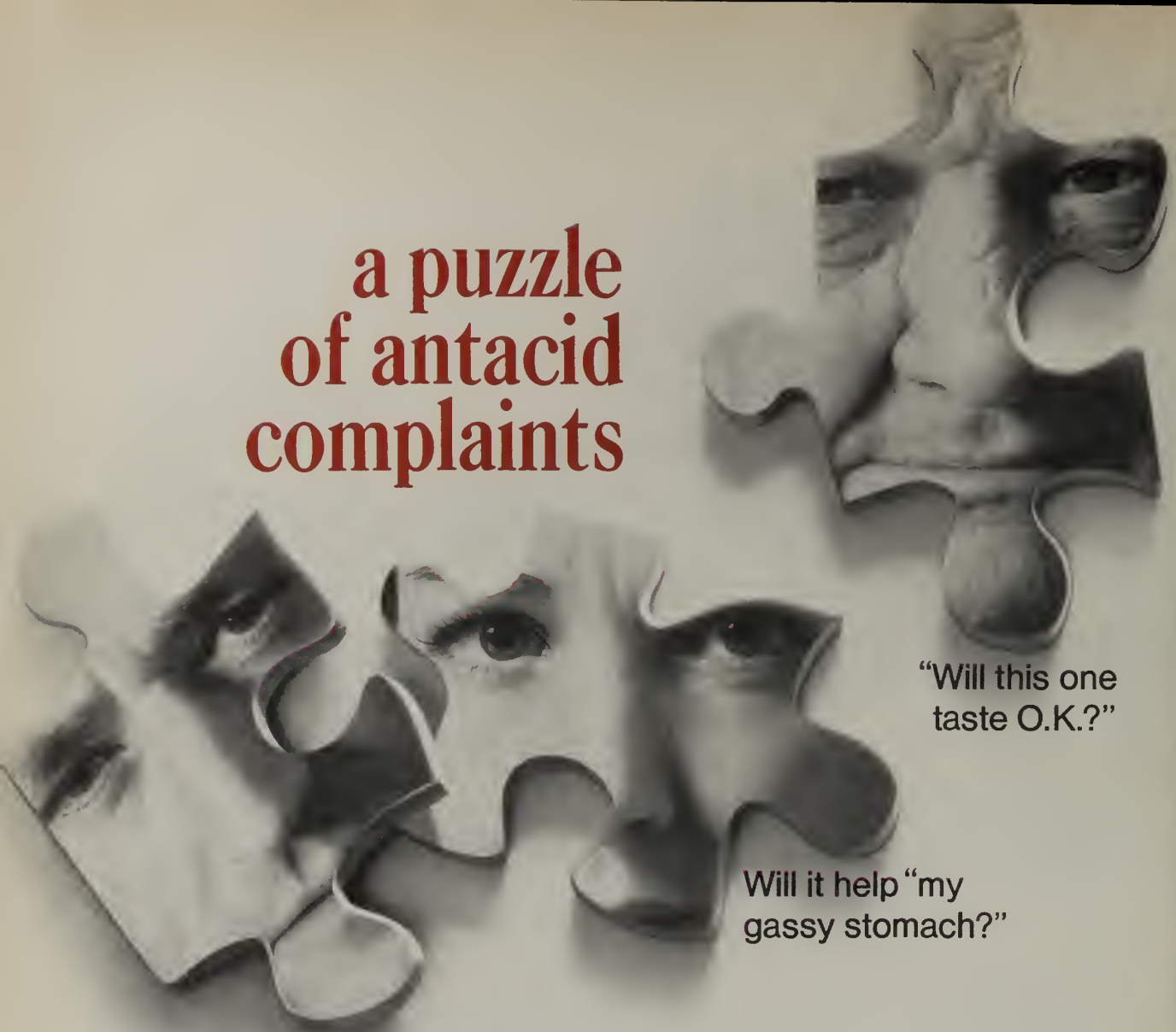
In men: When trichomonads are demonstrated, one 250-mg. oral tablet twice daily for ten days in conjunction with treatment of his female partner.

Dosage Forms: Oral tablets—250 mg.
Vaginal inserts—500 mg.

1. Complete list of references on request.

SEARLE

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Public Health....

MACK I. SHANHOLTZ, M.D.
State Health Commissioner of Virginia

Impact of the 1967-68 Influenza Epidemic

A total of 4,034 deaths were reported for Virginia residents in January 1968, substantially more than in any month since 1960 when monthly data were first tabulated. This January figure includes 3,836 deaths which actually occurred in that month plus 198 deaths which occurred prior to that time but were recorded with the January report. This large increase in deaths is primarily attributed to the widespread influenza epidemic accompanied by the extremely cold January temperatures.

As a rule the daily average of deaths by months varies considerably throughout the year with the winter months contributing the greatest numbers of deaths. These daily death averages for the past three non-epidemic years (1965-1967) ranged from 94 per day in August to 110 in December. The annual average for that period was 102 deaths per day. The deaths of the January 1968 report, however, represent an average of 130 deaths for each day of the month. That is 20 deaths per day greater than any monthly average of the previous three years. Since 1960, only February of 1963 with 135 deaths per day experienced a higher daily death average. The previous influenza epidemic was rampant at that time.

AVERAGE NUMBER OF DEATHS PER DAY BY MONTH
VIRGINIA, 1965-1967

Total	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
102	105	109	108	104	100	99	96	94	95	100	102	110

Studying death from influenza as the underlying cause does not indicate the full impact of the epidemic upon the population. In addition to directly causing death, it is thought that influenza precipitates deaths in older persons weakened from already existing serious chronic conditions. Pneumonia, too, appears much more frequently on death records during influenza epidemic periods; however, in many instances pneumonia, like influenza, is considered contributory rather than the underlying cause of death. While deaths attributed to influenza and pneumonia did increase considerably in January 1968 from the average of the previous three Januarys, 3.29 to 7.12 per 100,000 population, large increases were also recorded for other causes of death. Below are shown principal causes of death for January 1968 compared to the average of the previous three Januarys, 1965-1967.

Rates for the diseases of the cardiovascular system exceeded the previous three year average by 17.9 percent. Cirrhosis of the liver and diabetes were up a very high 56.3 and 47.8 percent, respectively. Among external causes of deaths, accident and suicide rates were considerably increased, 29.1 and 14.0 percent, while a 9.1 percent decline occurred for homicides. Very high death counts from falls and motor vehicles were chiefly responsible for the rise in accidental deaths. On the other hand, death rates of diseases of early infancy and congenital malformations were greatly reduced when compared to averages of the previous

three Januarys. This is probably the result of the substantial reduction in the numbers of births during recent years.

When considering age at death, the percentage of persons dying who were at least 80 years of age was markedly higher for the January 1968 deaths, 25 percent, when compared to the most recent death information available by age. The annual percentage for this age group usually lies around 20 percent. Monthly age variances cannot be considered here, however, since no other monthly data by age are available. The trend of fewer deaths under one year of age continues, following the previously mentioned declining number of births.

PRINCIPAL CAUSES OF DEATH WITH RATES PER 100,000 POPULATION AND PERCENTAGE CHANGE OF RATES
VIRGINIA, JANUARY AVERAGE 1965-1967 AND JANUARY 1968

Causes of Death	January Average 1965-1967		January 1968		Percent Change
	Number	Rate	Number	Rate	
Total	3,257	71.96	4,034	87.62	+21.8
Cardiovascular diseases	1,780	39.33	2,134	46.37	+17.9
Heart disease	1,266	27.97	1,499	32.56	+16.4
Cerebral vascular lesions	383	8.46	448	9.73	+15.0
General arteriosclerosis	67	1.48	89	1.93	+30.4
Other hypertensive disease	16	.35	30	.65	+85.7
Other circulatory disease	48	1.06	68	1.48	+39.6
Malignant neoplasms	468	10.34	546	11.86	+14.7
Influenza and pneumonia	149	3.29	328	7.12	+116.4
Accidents	182	4.02	239	5.19	+29.1
Motor vehicle	67	1.48	87	1.89	+27.7
Home	53	1.17	73	1.59	+35.9
Diseases of early infancy	102	2.25	85	1.85	−17.8
Diabetes	51	1.13	77	1.67	+47.8
Cirrhosis of the liver	32	.71	51	1.11	+56.3
Suicide	39	.86	45	.98	+14.0
Congenital malformations	37	.82	29	.63	−23.2
Homicide	25	.55	23	.50	+ 9.1
Chronic nephritis and renal sclerosis	22	.49	21	.46	− 6.1
Infections of the kidney	22	.49	21	.46	− 6.1
Tuberculosis, all types	16	.35	20	.43	+22.9
Hernia and intestinal obstruction	14	.31	16	.35	+12.9
Ulcer of stomach and duodenum	15	.33	15	.33	.....
Gastritis, enteritis, etc.	14	.31	9	.20	−35.5
All other causes	289	6.39	375	8.15	+27.5

PERCENTAGE DISTRIBUTION OF DEATHS BY AGE
VIRGINIA, 1964-1966 AND JANUARY 1968

	Total	Under 1	1-19	20-34	35-49	50-64	65-79	80+
Jan. 1968	100.0	3.8	2.3	2.5	8.4	22.3	35.5	25.2
1966	100.0	5.7	3.0	3.4	9.5	23.3	34.5	20.6
1965	100.0	6.4	3.1	3.5	9.2	22.9	34.5	20.4
1964	100.0	7.4	3.1	3.1	9.8	22.8	34.0	19.8

Mental Health . . . .

HIRAM W. DAVIS, M.D.

The Virginia Community Mental Health and Mental Retardation Services Act

The 1968 General Assembly was an extremely productive session with regard to mental health and mental retardation legislation. One of the primary objectives of the Department of Mental Hygiene and Hospitals, as recommended by the Virginia Mental Health Study Commission in 1965, favorably acted upon by the Assembly was a revision and recodification of Title 37 of the Code of Virginia. Future articles in this publication will deal at length with specific changes in commitment procedures and other aspects of the revision.

Because of the interest evidenced by those persons and organizations involved in the development of community services, and to provide information on a new aspect of such services included in the new Title 37, this article reproduces in its entirety Chapter 10 thereof dealing with Community Mental Health and Mental Retardation Services. This, as well as the entire revision, will become effective on October 1, 1968.

While no funds were made available for the 1968-70 biennium to implement the provisions of Chapter 10, it is hoped that those communities involved in planning comprehensive services will utilize the procedures outlined in Chapter 10 and implementing Regulations to be issued by the Department. Thus, local program planning and budgeting may be included in recommendations for funds to the Governor and the 1970 General Assembly.

It should be noted that this new legislation does not obviate the utilization of the present mental hygiene clinic program which continues to be authorized under the new legislation. It is intended to broaden the base of local services eligible for state participation and to provide a greater degree of local operation and control over such services.

There follows the text of Chapter 10 of Revised Title 37 of the Statutes of Virginia which will, as stated, become effective October 1, 1968:

STATUTES OF VIRGINIA—TITLE 37—CHAPTER 10 (Effective Oct. 1, 1968)

COMMUNITY MENTAL HEALTH AND MENTAL RETARDATION SERVICES

37.1-194. Purpose; Services to be provided.—The Department, for the purposes of establishing, maintaining, and promoting the development of mental health and mental retardation services in the State, may make matching grants to assist any county having a population of approximately fifty thousand or more or any city having a population of approximately seventy-five thousand or more, or any combination of counties or counties and cities having a combined population of approximately fifty thousand or more, in the establishment and operation of local mental health programs to provide:

(a) Collaborative and co-operative services with public health and other groups for programs of prevention of mental illness, other psychiatric disabilities, and mental retardation.

(b) Informational and education services to the general public, and lay and professional groups.

(c) Consultative services to courts, public schools, health and welfare agencies available to the public.

(d) Outpatient diagnostic and treatment services.

(e) Rehabilitative services for patients suffering from mental or emotional disorders, other psychiatric conditions and mental retardation.

(f) Inpatient diagnostic and treatment services.

(g) Research and training of personnel.

(h) Aftercare for the patient released from a mental hospital.

37.1-195. Community Mental Health Services Board; Appointment, and Membership.—Every city,

DAVIS, HIRAM W., M.D., *Commissioner, Department of Mental Hygiene and Hospitals.*

county or combination of counties or counties and cities establishing a community mental health services program, before it may come within the provisions of this act, shall establish a single community mental health services board, with neither less than five nor more than fifteen members. When any city or county singly establishes a program, the board shall be appointed by the mayor of the city or by the chairman of the county board of supervisors. When any combination of counties or counties and cities establishes a community mental health services program, the chairman of the board of supervisors of each county in the case of counties or mayor in the case of cities shall establish the size of the board between five and fifteen members, and shall select and appoint the members of said board.

37.1-196. Same; Term, Vacancies, Removal.—The term of office of each member of the community mental health board shall be for three years from the first day of January of the year of appointment, except that of the members first appointed, several shall be appointed for terms of one year each, several for terms of two years each, and the remaining members of the board for terms of three years each. The selection of members for one, two, and three year terms shall be as nearly equal as possible with regard to the total number of members on the board. Vacancies shall be filled for unexpired terms in the same manner as original appointments. Any member of a board may be removed by the appointing authority for cause, after being given a written statement of the causes and an opportunity to be heard thereon.

37.1-197. Same; Powers and Duties.—Every community mental health board shall:

(a) Review and evaluate community mental health and mental retardation services and facilities, both public and private, available to serve the community.

(b) Submit to the governing body or bodies of each political subdivision, of which it is an agency, a program of community mental health and mental retardation services and facilities.

(c) Within amounts appropriated thereon, execute such program and maintain such services as may be authorized under such appropriations.

(d) Enter into contracts for rendition or operation of services or facilities.

(e) Make rules or regulations concerning the rendition or operation of services and facilities under its direction or supervision, subject to applicable standards or regulations of the Department.

(f) Appoint a director of community mental health services whose qualifications meet the standards fixed by the Department and prescribe his duties. The compensation of such director shall be fixed by the board within the amounts made available by appropriation therefor.

(g) Prescribe a reasonable schedule of fees for services provided by personnel or facilities under the jurisdiction or supervision of the board and for the manner of collection of the same; provided, however, that all collected fees shall be deposited with the treasurer of the political subdivision of which the board is an agency, or, in the case of a joint board, with the treasurer of the political subdivision specified by agreement; provided further, that such collected fees shall be used only for community mental health purposes.

(h) Accept or refuse gifts, donations, bequests or grants of money or property from any source and utilize the same as authorized by the governing body or bodies of the political subdivision or subdivisions of which it is an agency.

(i) Seek and accept funds through Federal grants.

37.1-198. Mental Health Programs; Approval of Plan and Budget; Application for Grant.—Any city, county or combination of counties or counties and cities which establishes a community mental health board administering a mental health services program may apply for the assistance as provided in this act by submitting annually to the Department its plan and budget for the next fiscal year together with the recommendations of the community mental health board thereon. No program shall be eligible for a grant hereunder unless its plan and budget have been approved by the Department.

37.1-199. Same; Allocation of Funds by Department; Withdrawal.—At the beginning of each fiscal year the Department may allocate available funds to the community mental health and mental retardation programs for disbursement during the fiscal year in accordance with such approved plans and budgets. From time to time during the fiscal year, the Department shall review the budgets and expenditures of the various programs and if funds are not needed for a program to which they were allocated, it may withdraw such funds as are unencumbered, after reasonable notice and opportunity for hearing, and reallocate them to other programs. It may withdraw funds from any program which is not

being administered in accordance with its approved plan and budget.

37.1-200. Same; Withdrawal of County or City; State Matching Grants.—No county or city participating in a joint community mental health program shall withdraw therefrom without two years' notice to the other participating counties or cities unless the other counties or cities consent to an earlier withdrawal. State matching grants for any program shall be fifty percent of the total local expenditures, less collected fees, for salaries, contract facilities and services; operation, maintenance and service costs; travel expense of members of community mental health boards and other expenditures specifically approved and authorized by the Department.

37.1-201. Same; Construction and Mental Health Services Grants.—The State may grant up to fifty percent of local expenditures for construction of facilities to be used in community mental health programs. Grants may be made for expenditures for mental health services whether such services are provided by operation of a local facility or through contract with other public or private agencies.

37.1-202. Same; Staff Grants.—The State may grant up to seventy-five percent of local expenditures for staff members in community mental health programs.

Accidental Deaths Drop Slightly in 1967

Accidents caused an estimated 112,000 deaths in the United States during 1967, or about 1,000 less than in 1966, with the accident death rate dropping to a little below 57 per 100,000 population. Nevertheless, this rate was higher than in any year during the previous decade except 1966 when it reached 57.7.

The decline in accident fatalities in the last twelve months reflected in large measure a reduced toll from injuries in and about the home. A little over 28,500 persons sustained fatal injuries in such mishaps, a decrease of nearly 1,000 from the number in 1966.

Injuries arising out of and in the course of employment caused slightly fewer deaths than in 1966 when the total was 14,500; approximately 3,000 of the 1967 deaths were caused by motor vehicle accidents and were also included in the 53,000 motor vehicle fatalities referred to above. Public accidents other than those involving motor vehicles resulted in about 19,500 fatalities, or about the same as in 1966.

According to records maintained by the Statistical Bureau of the Metropolitan Life Insurance Company, nearly 1,300 persons were fatally injured during 1967 in catastrophic accidents, that is, accidents taking five or more lives. This is a more favorable record than the annual average for the past decade in the United States (excluding Alaska and Hawaii).

There were nine major catastrophes during 1967, each of which took at least 25 lives, and together they resulted in over 400 fatalities. Five of these disasters involved accidents in civil aviation, the most serious of which occurred on July 19 near Hendersonville, North Carolina, producing a death toll of 82 lives. The second heaviest loss of life was that in the November 20 crash of a scheduled airliner near the Cincinnati, Ohio airport, which took 69 lives. Tornadoes devastated several areas in northeastern Illinois on April 21, accounting for 55 fatalities, and a suspension bridge between Pt. Pleasant, West Virginia, and Kanauga, Ohio, collapsed taking 46 lives (including those missing). Thirty-eight persons died in a fire which swept a prison camp barracks at Berrydale, Florida, and 25 lost their lives in a fire which occurred in a penthouse restaurant at Montgomery, Alabama.

Motor vehicle and civil aviation accidents each accounted for about 30 percent of the catastrophic deaths in 1967. Fires and explosions, mostly in private homes and apartments, caused more than one fifth of the total. Military aviation and natural catastrophes were responsible for the major portion of the remainder. (Statistical Bulletin, Metropolitan Life Insurance Company)

Woman's Auxiliary....

Plan Your Schedule for the 1968 A.M.A. Auxiliary Convention—San Francisco—June 16 to 20:

Friday, June 14:

Finance and Communications Committees meeting. Joint meeting with Program Development Committee.

Saturday, June 15:

Meeting of the Board of Directors. National chairmen and National representatives to meetings (auxiliary and others) are invited to attend the afternoon meeting to give brief reports.

Board of Directors Luncheon. Guests will be National Board, National Chairmen, Past Presidents and Honorary members.

Sunday, June 16:

Meeting of the President-elect, Mrs. C. C. Long, with the 1968-69 Chairmen; Bylaws Committee meeting; Resolutions Committee meeting. Registration and Hospitality Room will open.

Coffee will be served until 1:00 p.m. Reception, honoring the National President and President-elect, will be held at the St. Francis Hotel. "Dutch treat" bar with hors d'oeuvres courtesy of National Auxiliary. Members, husbands and guests cordially invited.

Opening Meeting of the AMA House of Delegates.

AMA Medicine and Religion Program, California Masonic Memorial Temple.

Monday, June 17:

Formal opening of the convention. Business meeting.

Guest Day Luncheon for leaders of National Women's Volunteer Organizations, Auxiliary members and guests. Guest speaker: Dr. Henrik L. Blum, Clinical Professor of Community Health Planning, University of California.

The afternoon program will schedule part of the state reports under the direction of the National Vice Presidents: remainder on Tuesday morning.

NOTE: Nominations for the 1969 nominating committee will be made during the business meeting Monday morning. The election will take place Tuesday afternoon in a polling place so designated. All delegates must be supplied with the proper credentials.

Tuesday, June 18:

Presentation of state reports (continued from Monday).

Annual luncheon, honoring National Past Presidents. Guest speaker: Dr. Milford O. Rouse, President, American Medical Association.

COMMUNICATIONS PROGRAM. The Communications Committee will present a program with emphasis on "How to get the most out of your State Newsletter" and suggestions to state presidents, presidents-elect and state editors on the values of the newsletter in public relations activities.

There will be a presentation of health education films available through AMA Film Library and other sources for use by county and state auxiliaries.

Wednesday, June 19:

General Meeting. Amendments, resolutions, reports and installation of the new officers will follow.

Board of Directors luncheon and meeting with 1968-69 Chairmen.

Postconvention Conference for Presidents and Presidents-elect (open to all Auxiliary members).

Presentation and discussion of 1968-69 programs and projects.

AMA Round-Up—Ernest B. Howard, M.D., Assistant Executive Vice-President, American Medical Association.

Thursday, June 20:

Meeting of the Board of Directors. Leadership Conference for Workshop Leaders.

A program for Pre-teens and Teen-agers of Auxiliary members is planned. Complete details appear in the May Issue of MD's WIFE.

Norfolk Chapter on the Move in Many Directions—Mrs. Theodore Adler and Mrs. Frank E. Rowell are Co-Chairmen of the two-day State Rehabilitation Meeting to be held in Williamsburg on November 3rd and 4th—so mark your calendars now. They will have an outstanding panel of doctors, covering all phases of medical rehabilitation. A workshop will also be conducted . . . Mrs. Charles Sale, Community Health Chairman, has really done a bang-up job this year. Shoes have been collected for the Goodwill, where the handicapped repair, resole, reheel and sell them. She also was busy collecting old hats and hat boxes for Eastern State Hospital where they were redecorated by the patients. The project was carried into the forefront by door prizes wrapped with miniature shoes or hats on each. . . . Mrs. Aubrey Shelton has just about the cleverest and certainly the most glamorous way of raising funds for A.M.A.E.R.F. with professional make-up sessions plus a 10% discount at several shops. Doesn't that sound gorgeous! . . . Norfolk girls also held a get-acquainted coffee for their new members with their Board; sponsored two lovely teas for the nursing students; and sponsor Future Nurse Clubs at five high schools. On Doctor's Day they held a black tie dinner dance and the word is that it was the loveliest ever!. . . . Not the least of their fine projects is their Museum Project . . . a medical display contrasting advances in medicine through various wars from the Civil War to Viet Nam. Their medical display was also set up in one of the department store windows during National Hospital Week and is now shown to High School groups interested in Health Careers. . . . Hats off to Norfolk and a job well done!

The Auxiliary Was Represented at Two Recent Meetings of importance to all of us. Mrs. Robert D. Keeling attended the annual meeting of the Virginia Council on Health and Medical Care in Richmond. Mr. Caesar Branchini, Associate Director, Community Health and Health Education of the Equitable Life Assurance Society spoke on putting meaning into partnership for health. He discussed the planning and organization of facilities and services, recruitment and training of health manpower, provision of quality care, and the cost and financing of such care—all on a partnership basis. The job is a challenge and a responsibility of health groups everywhere concerned with medical care. . . . Mrs. Reuben F. Simms attended the 12th Annual Nutrition Forum presented by the Virginia Council on Health and Medical Care and Thalhimers on April 22nd and 23rd. The theme of the forum was "Nutrition Faces the Food Revolution". She reports there is a growing concern that we are not receiving the proper nutrition in this country. It was pointed out that this has become a political issue—and should be . . . malnutrition, hunger and starvation exists on a large scale basis according to several studies presented at the meeting. It was urged that we start teaching better nutrition to everyone beginning at the grade school level and improve attitudes to nutritional foods. They stressed that it is the responsibility of every homemaker, restauranteer and grocer.

Help Wanted Overseas . . . . A challenging opportunity comes to us from International Health. (Mrs. Joseph M. Straughan is our State Chairman.) Medical services are desperately needed overseas in developing countries. You and your husband can realize a challenging and exciting opportunity by sharing in such a great cause. Wives do not have to be trained in a paramedical field to be of value—just be willing and able. Countries in desperate need include Vietnam, Hong Kong, Mexico, Guatemala, Afghanistan, Algeria, Honduras, Malaysia, Nigeria, Tunisia, Haiti, Laos, Thailand and many other countries in Africa, South and Central America, the Middle East, Far East and Southeast Asia. For details contact the International Physician's Placement Service, American Medical Association, 535 North Dearborn Street, Chicago, Illinois 60610.

Let's Reminisce!

Medicine in a noble and honorable science, and its votaries ought to be honorable men; but it is a lamentable fact, that in this our day, there are many, who, for the sake of gain, will stoop to anything, not matter how contrary to the rules of the profession; who peddle out their practice like the Jew his merchandize, for as low as one-tenth its real value, merely for the sake of custom.

The following will exemplify this:

There are in my immediate vicinity two or three physicians, who make it their business to go about trying to engage families or individuals by the year. If they cannot get them for one price, they will for another. Their object is to get the practice, no matter how unfair or unprofessional the means. They go to families and insist that they should employ them by *the year, as it will be so much cheaper for them to do so.* One of them says to those, who do not employ a physician in this way, *send for me any how* whenever any of you get sick, and pay me what you please. I am a young man. It does not take much to support me. He approaches a blacksmith with about four in family in all, and a wife, who expects to be confined in two or three months in this manner: "How much will you charge me for shoeing my horse by the year?" "Eight dollars," is the reply. "Well, (says the doctor) and I will practice in your family by the year for $3.50." The blacksmith things it certainly cheap enough, and engages him. This is the minimum price per family, and ten dollars is the maximum, so far as I have been able to learn. (Editorial, The Virginia Medical Journal, September 1856)

Editorial . . .

Infectious Mononucleosis—A Multifaceted Entity

AN ACCOUNT OF THE DISCOVERY, by Philadelphia research Drs. Werner and Gertrude Henle, of a clue to the etiological agent responsible for infectious mononucleosis, that appeared in this year's January issue of the Proceedings of the National Academy of Sciences, prompts the author of this editorial to record something of his experience with this disease whose cause for long had remained—and still does remain—a mystery. (The most claimed by the Drs. Henle was that they had implicated a herpes-type virus called EBV Virus as the "probable cause" of infectious mononucleosis.)

It was in 1889 that Dr. Emil Pfeiffer of Germany reported a group of cases presenting a symptom complex sufficiently applicable to the group as a whole to warrant the announcement of his discovery of a disease unique unto itself. For this malady Dr. Pfeiffer chose the term Drusenfieber, lymph gland fever. Enlarged cervival lymph glands, a reddened throat "without exudate" and an enlarged liver and spleen were listed by Dr. Pfeiffer as cardinal characteristics of Drusenfieber.

Thirty-one years later, i.e., in 1920, Sprunt and Evans reported a significant increase in the number of large mononuclear lymphocytes in blood smears of individuals presenting the clinical picture described by Pfeiffer. Shortly thereafter the syndromes defined by the foregoing pioneers in this field came to be accepted as one and the same.

In 1930, Glanzman, drawing upon observations made during a major epidemic in Berne, Switzerland, from a clinical and laboratory standpoint, not only confirmed the identity of the Pfeiffer and Sprunt-Evans syndromes, but further contended that a membranous exudate in the throat was entirely consistent with the diagnostic criteria of monocytic angina and glandular fever. Pfeiffer, 41 years earlier, had pointedly mentioned the absence of exudate in the throats of his series of cases.

At any rate, two more or less separate clinical groups appear to prevail, (1) The angiose or pharyngeal form, that is most common in young adults, and (2) The glandular or Pfeiffer type, more common in children.

There is no reason whatever to think that infectious mononucleosis did not exist long before it was recognized as a disease sui generis by Dr. Pfeiffer.

To the end of avoiding prolixity, a detailed review of the protean manifestations of infectious mononucleosis and/or a definitive consideration of its several forms will not be undertaken here. Complete descriptions of this multifacted disease entity are, of course, contained in numerous textbooks of medicine. However, it is interesting to this commentator to reflect that as a member of the Medical Corps of the U. S. Navy throughout an active duty span in excess of 33 years (1923-1956) at an assortment of the Navy's major hospitals and other Naval

Medical Department facilities, both within and beyond the continental limits of the United States, and aboard a number of ships in which he served, only an occasional case of mononucleosis is recalled.

The reasons for this rather extraordinary matter of fact are several: (1) Few, if any maladies, masquerade under so many different guises as infectious mononucleosis. (2) It was not until 1932 that the heterophile antibody test was introduced by Paul and Bunnell, and it wasn't until 1935 that that test, which depends upon the presence of sheep cell agglutinins in the patient's serum, was made more specific by Bailey Raffel and Davidsohn through the utilization of absorption principles. Therefore, it is highly probable that an incalculable number of cases of infectious mononucleosis were encountered along the way by this writer, but simply were not recognized as such. (3) Undoubtedly the fact too, that the specialty field of the author—insofar as specialization in the Navy is feasible—was general surgery, constitutes another reason for his limited dealing with mononucleosis prior to his retirement from active duty in the Naval Service.

The reality that in recent years infectious mononucleosis has come to acquire a rating high in the ledger of causes of man-days lost in the U. S. Navy and Marine Corps would appear worthy of special note: Over the past seven years the average number of active duty personnel in all categories of the Navy and Marine Corps has been in the order of 1,050,-000. Out of this total the number of cases of infectious mononucleosis has averaged 1,529 per year, and the average length of patient stay on the sick list due to this disease has been 29 days. This latter figure seems high; and it is high, but it should be borne in mind that the length of incapacitation of Armed Forces personnel for any cause is predicated upon the individual's being able to perform full duty when discharged from a medical facility. Hence the length of stay in an inactive duty status of Armed Services personnel far exceeds the length of time the same individual in civilian life, in the absence of complications, need necessarily be hospitalized, or kept inactive.

It also appears from the record that complications of infectious mononucleosis encountered in the Navy and Marine Corps have been fairly infrequent. In 1,589 cases of this malady occurring in these two branches of the Armed Forces during fiscal year 1967, complications ranging from streptococcal tonsillitis (the most common complication), to orchitis and epididymitis (the least common), were recorded in 116 cases or (if time has not rendered this writer's never better than puny aptitude for arithmetic wholly nul and void), about 7% of the total. Ten cases of infectious hepatitis; seven of disorders of the blood and blood forming mechanism (thrombocytopenia, hemolytic anemias, etc.); two of polyneuritis; one of stomatitis and one of testicular involvement were reported in this 1967 series.

Reference to the Navy's statistical records does reveal one seeming peculiarity relative to infectious mononucleosis believed not to have been generally recognized, and that is the apparent predilection of this disease for the Caucasian in contrast to the Negro race. Of the 1,655 cases of

infectious mononucleosis occurring in the Navy and Marine Corps active duty personnel during fiscal year 1966, only 27, or 1.6% were reported in the Negro race; whereas 5.5% of active duty Navy and Marine Corps personnel were negroid.

Throughout a period, now approaching twelve years, during which this commentator has served as University Physician at one of America's top flight institutions of learning whose annual enrollment has been in the order of 15,000 students of both sexes, ranging in age from 17 to 70 years, and with one or more representatives of virtually every civilized nationality on earth, the incidence of infectious mononucleosis has been second only to upper respiratory disorders, such as common colds; gastro-intestinal upsets and minor injuries. And no health condition has caused so extraordinary a measure and so diverse a character of concern among the students by and large as has infectious mononucleosis or—in the idiom of the students—"mono".

Why this is so poses an interesting proposition and educes such terms as baleful, benign, bugbear and beneficence. Among the approximately 1,000 bona fide cases of infectious mononucleosis encountered by the author within his student clientele over an eleven and one half year period there have been individuals to whom each of the four foregoing terms would have been apposite.

Baleful as a descriptive term has been applicable to the symptom complex and morbidity manifested by about 10% of the students comprising the encompassed series. Severe and antibiotic resistant tonsillitis with exudate resembling that of diphtheria has been common to most in this category. Two or three degrees of temperature elevation has been the rule at the onset, as has been enlargement of the cervical lymph nodes—in both the anterior and posterior chains, but particularly anteriorly. Occasionally this cervical lymphadenopathy has been quite marked, but never approaching that seen in lymphatic leukemia or Hodgkin's disease. These enlarged lymph glands have never been more than moderately tender. Palpable axillary and inguinal glands have constituted a part of the diagnostic picture in probably one third of the sum total of the students whose heterophile antibody tests have been positive. Enlargement of the spleen in these cases has not infrequently been demonstrable, but demonstrable enlargement of the liver has been rare. A maculopapular skin rash has been observed in only a few cases. There has not appeared to be any consistent correlation between the splenic and/or liver enlargement and the student's subjective symptoms, nor has there been much, if any, discernible relationship between the grade of invalidism manifested and the degree of serological positivity in a given patient. The average duration of the acute phase of mononucleosis in the students comprising this observed group has been about a week. The period of convalescence however, has, in the main, been more in the nature of a drag than of a rebound. This facet of the disease has varied rather widely, depending largely upon the inherent mettle of the patient. A recrudescence of acute symptoms has seldom been observed in this series. Hospitalization has rarely been necessary. A few have dropped out of school for the re-

mainder of a term and have gone to their homes. A majority of these, in due course, have returned to school and not uncommonly have reported that they were kept in bed at home for six weeks, but upon what justification few have known.

Benign is the qualifier appropriate to the grade of mononucleosis that has afflicted 90% of the student group under consideration. Along with or aside from the more conventional subjective symptoms included in various sources of reference, a complaint of extreme lassitude, without patent reason, has, in this writer's view, been a cardinal symptom sufficiently often to warrant its inclusion among the leading diagnostic criteria of infectious mononucleosis.

It should be mentioned that infectious mononucleosis would undoubtedly have gone undiagnosed in many belonging to this "benign" category, but for the fact that, for a considerable span of months, specimens of blood on all potential cases were sent to the National Institutes of Health and to the Naval Medical Research Institute at Bethesda as contributions to research programs being conducted at those centers and where heterophile determinations were made, gratis. In any event, a positive heterophile antibody titer of 1:56 or higher, preabsorption, or of a 1:28, but at least of 1:14, postabsorption, performed in a certified hematology laboratory, has been a sine qua non to a diagnosis in this entire group of cases.

Upon the score of complications in this student series of 1,000 cases of infectious mononucleosis, the interjection of brief supplementary comment is thought to be appropriate. It is realized that evidence of certain complications incident to the infiltration of deranged blood cells and the virus of mononucleosis into various organs of the body might have been revealed by highly specialized laboratory tests to which it was not expedient to subject this group of students. Some degree of hepatocellular damage might have been revealed by specific serological tests. These tests were foregone in the interest of avoiding time loss from classes and nonessential expense to the student. Neurological disorders, i.e. acute polyradiculopathy, encephalitis and meningeal involvement comprising the so-called Guillain-Barré syndrome, and claimed by researchers in this field to be common complications of infectious mononucleosis, simply have not been recognized unless possibly in mild, noninvaliding and self ameliorative forms. In other words, if such symptoms as general malaise, headache, musculoskeletal soreness and drowsiness associated with the commonly accepted signs of infectious mononucleosis (serologically confirmed) could have been ascribed to central nervous system involvement, they have been too transient in their presence and too spontaneous in their disappearance to warrant their being classed as indicative of the Guillain-Barré syndrome. Suffice it to say that in this series of definitely diagnosed cases of infectious mononucleosis in 1,000 (plus) college and university students, the mortality rate from the primary disease or any complication thereof has been zero.

The term, bugbear, in one sense, connotes the measure of concern experienced by students whose clinical picture has been indicative of

mononucleosis and whose diagnosis has been forthrightly serologically confirmed. In another sense however, a greater measure of concern has been manifested by many students who have professed exceeding anxiety lest they might be or, in fact, lest they might not be coming down with this "dreadful" malady. There has been that inevitable element comprising those who have appeared to be imbued with the idea that unless and until they had had "mono", they had not quite "arrived".

The bearing of the term, beneficence, is indirect. Here the situation usually has involved a student who, having taken his cue from the grasshopper rather than from the ant, has found himself academically in the "doghouse" and has been desperately in need of a way out. If he could manage to come up with the subjective symptoms and clinical picture of infectious mononucleosis and could "pull" a positive heterophile reaction, he has "had it made". He thus has been invested with an acceptable excuse for dropping out of school with no credit instead of discredit. So, once more, the adage having to do with the illness of a wind that blows no good to anybody would, indeed, have come to the fore.

H. Lamont Pugh, M.D.

The Future of the Local Medical Society

SEVERAL MONTHS AGO the Medical Society Magazine Group, a non-profit association of magazines published by county medical societies throughout the country, took the American Medical Association to task for "downgrading" American county medical societies. The editorial in question, which raised the ire of the magazine group, appeared in the January 29 issue of the AMA News, and said, in part, "Low, and declining attendance at many local society meetings seems to indicate that there has been a decline in the influence, prestige and effectiveness of the average local county medical society as a unit in medical organization." Whether we like it or not, it is all too true.

Attendance at the city, and probably at the county medical societies, has declined in Virginia. Parenthetically, as a result of a political quirk, the societies in all the cities of Virginia, Baltimore and St. Louis are listed as city medical societies; the remainder throughout the country are designated as county societies. The trend downward has continued for the past two decades. A number of factors are responsible; some were not mentioned by the AMA article or the Board of Trustees original report last fall on which the AMA editorial was based.

Unquestionably the many hospital staff and committee meetings have drained off much of the professional zeal that otherwise might be channeled to the local society gatherings. The specter of the inspector from the Joint Commission on Accreditation of Hospitals has prompted every hospital to set up the usual infection, tissue, bed occupancy and the other drab array of committees that must meet weekly, biweekly or monthly.

The general staff and the various specialty meetings are also held at regular and frequent intervals. If a community is served by several hospitals, the younger physician, especially, finds it beneficial to become identified with as many hospitals as possible. Each institution in turn takes its pound of flesh in the form of various time-consuming assignments. If this attendance falls below 50 percent in the designated categories his privileges are threatened.

The city or county society, meanwhile, mercifully makes no requirement concerning attendance on the harried physician, so he in turn, goes to his home on the night of the local meeting and spends the evening with his family. The physician, who formerly endeavored to keep abreast in his profession by attending his local medical meeting, now finds the hospital specialty and staff meetings to be of greater value. The many recently organized teaching and postgraduate courses place the local society at a further disadvantage. The result of all this is that the local meeting deals chiefly with questions concerning medical economics and deciding just who will represent the society at the next state meeting.

The AMA Board of Trustees' report throws a ray of hope for the harassed hospital physician. It states "The committee understands that it is *not* a requirement of the Joint Commission on Accreditation of Hospitals that meetings relating to hospital staff affairs be intramural; that JCAH has no interest in locale so long as the function is performed and satisfactorily recorded. Opposition to extramural meetings might be expected to come from hospital administration. Intramural meetings have some possibility of domination by non-medical individuals." Thus far virtually no advantage has been taken of joint meetings by the local societies or by various hospitals on a rotating basis. The physicians have become so browbeaten by the numerous rules from various sources that they may fear this is just another ruse. This matter should be clarified for it would ease the burden for the hospital-ridden physician and the local medical society would be correspondingly benefited. (Ed.: Dr. Robert E. Mitchell, Jr., is chairman of a committee currently looking into this matter. A report will be made by him in an early issue of the VMM.)

These thoughts were occasioned by a recent suggestion that the Academy of Medicine building in down-town Richmond be discarded and a new headquarters be located in the west end of the city. The chief argument advanced in favor of this move was the possibility of a better attendance. If the experience in other parts of the country has any bearing on our local problem a change of location will be of little aid. The question is not one of geography but how an additional two hours can be fitted into the already crowded schedule of the busy physician.

H. J. W.

News ....

Calendar of Events

American Medical Association—Annual Meeting—San Francisco, California—June 16-20, 1968.

Tri-State Medical Society—Annual Meeting—The Carolinian—Nags Head, North Carolina—June 16-18, 1968.

Southern Association of Workmen's Compensation Administrators—Annual Convention—Statler Hilton Inn—Williamsburg—June 16-20, 1968—All Society members invited.

Department of Psychiatry and Continuing Education Program, University of Virginia—Seminar for general practitioners entitled "Psychiatric Aspects of Family Practice"—Sheraton-Alexandria Motor Inn—Alexandria—June 21-22, 1968.

Seaboard Medical Association—Annual Meeting—Nags Head, North Carolina—June 21-23, 1968.

Medical Aspects of Sports—Sponsored jointly by The Medical Society of Virginia and Virginia High School League—University Hall—Charlottesville—July 21, 1968.

Health Executives Workshop—Sponsored by Virginia Council on Health and Medical Care—Hotel Roanoke—August 28-30, 1968.

9th Annual Cardiovascular Symposium—Sponsored by Tidewater Heart Association and Council on Clinical Cardiology, American Heart Association—Lake Wright Motel, Virginia Beach—September 6-7, 1968.

The Medical Society of Virginia—Annual Meeting—Hotel Roanoke—October 13-16, 1968.

Virginia Rehabilitation Association—Annual Meeting—Williamsburg Lodge—Williamsburg—November 3, 1968.

Southern Medical Association—Annual Meeting—New Orleans, Louisiana—November 18-21, 1968.

New Members.

The following new members were received into The Medical Society of Virginia during the month of April:

Gerardo Bellodas, M.D., Richlands

Lawrence Albert Bernert, Jr., M.D., Portsmouth

Walter Bjarnesen, M.D., Shenandoah

Gloria Godbey Brennan, M.D., Fairfax

Robert Hueh Brumfield, Jr., M.D., Roanoke

William James Crowley, M.D., Charlottesville

Allen Stiles Gardner, M.D., Springfield

David Rollo Hawkins, M.D., Charlottesville

Clarence Adrian Holland, M.D., Virginia Beach

Lawrence Delano Kelley, M.D., Martinsville
John Bradley LaFave, M.D., Martinsville
Clara Jane Toothman, M.D., Coeburn
Anastasios Tony Tsitos, M.D., Falls Church
Peter Wadewitz, M.D., Petersburg
Delosa Anthony Young, M.D., Springfield

Virginia Academy of General Practice.

At the annual meeting of the Academy, held at the Chamberlin, Fort Monroe, May 9-12, Dr. Howard I. Kruger, Norfolk, was installed as president, succeeding Dr. J. Powell Anderson, Waynesboro. Other officers elected are: Dr. A. Epes Harris, Jr., Blackstone, president-elect; Dr. Clarence W. Taylor, Jr., Shawsville, vice-president; Dr. Alan Mackintosh, Vienna, secretary; and Dr. Jose D. Coll, Richmond, treasurer. Directors to serve three year terms are Drs. Levi W. Hulley, Jr., Richmond; Thomas H. Jennings, Jr., Bedford; Walter S. Barton, Wytheville; and Wallace E. Baker, Springfield. Dr. R. G. McAllister, Richmond, was elected delegate to the American Academy of General Practice Congress.

Loudoun County Medical Society.

Dr. William P. Frazer, Purcellville, has been elected president of this Society, with Dr. James L. Towe, also of Purcellville, secretary-treasurer.

Dr. Thomas W. Murrell, Jr.,

Richmond, has been elected a member of the Board of Directors of the American Dermatological Association for a term of six years.

First Citizen.

Dr. Robert M. Campbell has been named First Citizen of Portsmouth. The award is sponsored annually by Lodge 898, Loyal Order of Moose. Dr. Campbell has practiced surgery in Portsmouth for the past fifteen years and always finds time to play a leading role in community betterment programs.

Dr. Braxton Honored.

Dr. Herman H. Braxton, Chase City, has been presented with the Citizenship Award by the Jaycees in appreciation of his thirty-four years of unselfish service to Chase City and the surrounding areas. This is one of the highest annual awards made by the Jaycees.

Initiates into Honorary Medical Society.

At the annual spring lecture of the University of Virginia Chapter of Alpha Omega Alpha, a national honorary medical society, three physicians and three medical students were received into membership. The physician members of the faculty of the University are Dr. William G. Thurman and Dr. Daniel N. Mohler. Dr. Paul D. Camp, Richmond, is the other new member.

Dr. J. R. Freeman, Jr.,

Who has practiced in Cape Charles for the past eighteen years, has moved to Richmond where he will be associated with the Richmond Memorial Hospital in the emergency service.

Dr. Charles M. Caravati,

Medical College of Virginia, Richmond, delivered the George M. Boyer, M.D. Fund Lecture at the annual meeting of the Medical and Chirurgical Faculty of the State of Maryland in Baltimore on April 17th.

Dr. Caravati and Dr. George P. Vennard addressed the Alexandria Medical Society on April 10th on the subject of Hepatitis.

Dr. Caravati was also inducted as a Master in the American College of Physicians at its annual meeting in Boston.

Dr. Julian R. Beckwith, Sr.,

University of Virginia, Charlottesville, has been re-elected to serve a three year

term on the Board of Directors of the American College of Physicians.

Dr. Glendy Retires.

Dr. R. Earle Glendy, Roanoke, has announced his retirement from the active practice of medicine. He has accepted the post of Chief of Cardiology at the Roanoke Memorial Hospital. His new work will also involve supervision of the Intensive Care Unit and certain teaching duties.

Dr. John A. Gill,

Richmond, was a participant in a symposium on medico-legal matters at the annual meeting of the American Academy of Facial Plastic and Reconstructive Surgery in Hollywood Beach, Florida, April 27-30.

The Virginia Guild for Infant Survival.

Recently a chapter of the Guild for Infant Survival was established for the State of Virginia. The Guild is interested in all aspects of crib death which includes education, investigation and the support of research in this field. Interested persons may obtain information by contacting Mr. John Dalby, President, 8346 Carnegie Drive, Vienna, Virginia 22180.

Home-Office for Sale.

In exclusive Chevy Chase, Washington, D. C. One block off Connecticut Avenue at Chevy Chase Circle. Six room office suite, 4 bedrooms, formal living and dining rooms, etc. Call owner (202) 244-3327. (*Adv.*)

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Profexray, 15 MA, x-ray unit, complete with tilt table and fluoroscope. Suitable for office use. Also a stainless steel developing tank. Call owner at (703) 889-1224. (*Adv.*)

Hospital Physicians Wanted.

The Medical Staff, Board of Directors, and Administration of Winchester Memorial Hospital, Winchester, Virginia, are agreed to a plan of four full-time physicians to be in private general practice limited strictly to the Emergency Department of the Hospital. Average work week is forty-two hours. A generous guarantee has been established.

Four experienced physicians having a Virginia license are needed. The target date is July 1, 1968. Interested physicians contact the Committee Chairman, James A. Miller, M.D., Box 554, Winchester, Virginia. Telephone 662-0377 (area code 703). (*Adv.*)

For Sale.

X-Ray Unit, G.E. 300 MA, 125 KVP. Complete and guaranteed in excellent condition. Call collect Mr. Grady or Mr. Finn, 275-9239, Hull Street Outlet, Inc., 3820 Jefferson Davis Highway, Richmond, Virginia 23234. (*Adv.*)

Obituary . . . .

Dr. Wilkins Jeffress Ozlin,

South Hill, died April 23rd at the age of seventy-one. He was a graduate of the Medical College of Virginia in 1922 and had practiced in South Hill for thirty-five years. Dr. Ozlin had practiced in Dundas and was a member of the staff of Catawba Sanatorium before locating in South Hill. He served as chief of staff at Community Memorial Hospital from 1958 to 1962. Dr. Ozlin has served as president of the Fourth District Medical Society. He had been a member of The Medical Society of Virginia for forty-five years and served on the Council from 1948 through 1956. Dr. Ozlin was a past president of the South Hill Rotary Club and a veteran of World War I.

He is survived by his wife, a daughter, a sister and two brothers.

Dr. Cardwell Camden Nuckols,

South Hill, died April 22nd. He was forty-eight years of age and received his medical degree from the University of Virginia in 1943. Dr. Nuckols had practiced in South Hill since 1958 and served as chief of staff of the Community Memorial Hospital from 1962 until 1966. Prior to that time he had been at the University of Virginia and with the Army Medical Corps during World War II. Dr. Nuckols was past president of the South Hill Rotary Club. He had been a member of The Medical Society of Virginia since 1951.

His wife, a son and a daughter survive him.

Dr. Fears.

Dr. L. O. Fears entered VMI in September 1940 and graduated in 1944. He graduated from the University of Virginia School of Medicine in 1948. Upon graduation he interned at the University of Colorado Medical Center, Denver. He began a residency in OB-GYN at the University of Virginia, but because of illness it was necessary that his education be discontinued for a time. After a training period in the Public Health Service he went to Galax where he served as Health Officer until 1953 when he transferred to this area. Here he covered a four county area, later a five county area and the City of Winchester. Front Royal was his home from 1953 to 1961 when he moved to Winchester where he lived until his death. During his stay in Front Royal he attended Johns Hopkins University and earned a degree of Master of Public Health.

Dr. Fears was well known and loved by all the physicians in the Medical Society of Northern Virginia. He was particularly noted for his encouragement in the development of preventive medicine through any means at his disposal and was continually inspiring others to use their talents to the fullest. Under his leadership new Health Department buildings were erected in Luray and Winchester.

Therefore, Be It Resolved that the Medical Society of Northern Virginia and the Medical Profession has lost an honored and beloved member and that this Society record its deep sorrow at his passing. Also, that a copy of this resolution be kept in the Society files and a copy be sent to his family.

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SPOTSWOOD ROBINS, M.D.
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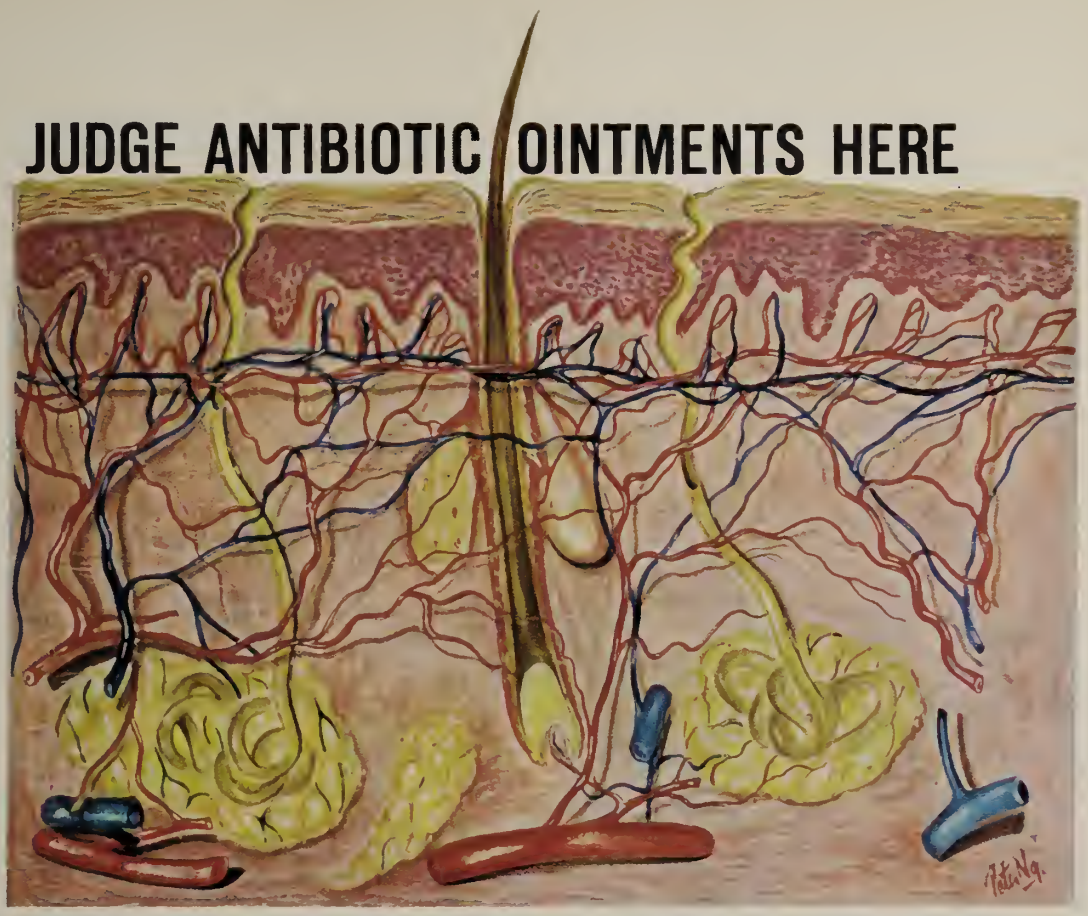
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Warning
Fatal cases of aplastic anemia have been reported following treatment with Tegretol. Agranulocytosis, thrombocytopenia and transitory leukopenia have also been observed.
Complete blood and platelet counts should be done prior to and at regular intervals during treatment with the drug (see recommendations under "Important Note and Precautions") to help in the early detection of serious bone marrow injury. Abnormalities in initial blood tests should rule out use of the drug. Also, patients should be made aware of such early toxic signs of a potential hematologic problem as fever, sore throat, mouth ulcers, easy bruising and petechial or purpuric hemorrhage. Should such signs appear, the patient should be advised to discontinue the drug and to report to the physician immediately.

Indication Tegretol is indicated in the treatment of the pain associated with true trigeminal neuralgia. It is not a simple analgesic and should never be administered for relief of trivial facial aches or pains.
Contraindication Do not use in those with a known sensitivity to any tricyclic compound or in those being treated with M.A.O.I. agents. As long a period as possible should elapse before using Tegretol in patients who have been treated with M.A.O.I.'s, with a minimum of 7 days. In such cases, initial dosage should be low and the patient's reaction to gradual increments closely observed.
Important Note and Precautions Familiarity with the clinical symptoms which lead to an accurate diagnosis of true trigeminal neuralgia and with the complete prescribing information, careful patient selection, a thorough examination before treatment and close patient supervision throughout the treatment period are essential to the safe and effective use of this drug.
Where feasible, Tegretol should not be used in conjunction with any potent drug which may increase the possibility of toxic reactions.
In pregnancy, the drug should not be prescribed during the first trimester and thereafter only to patients in whom the clinical situation warrants the potential risk. It should not be administered to nursing mothers.
Patients with increased intraocular pressure should be closely observed during treatment with this drug because of its anticholinergic effect.
Because of the drug's relationship to other tricyclic compounds, the possibility of activation of latent psychosis and, in the elderly, of confusion or agitation, should be considered.
Dizziness and drowsiness may occur and patients should be cautioned about the hazards of operating machinery or automobiles and of engaging in other hazardous tasks.
Use cautiously in patients with a history of coronary artery disease, organic heart disease, congestive failure or liver disease.
Before initiating therapy, the following laboratory procedures should be performed:
1. Complete blood and platelet counts which, if abnormal, should rule out the use of the drug.
2. Baseline evaluations of liver function.
During treatment with Tegretol, the following laboratory procedures should be performed:
1. Complete blood and platelet counts should be done at intervals of one week during the first month of drug treatment, every two weeks during the second and third months, and at monthly intervals thereafter for as long as the patient is taking the drug. A trend toward a decreasing white blood cell count should suggest a dosage reduction and more frequent laboratory and clinical evaluations. Should this trend continue, the drug should be discontinued.
2. Liver function tests must be performed at regular intervals during treatment with this drug since liver damage may occur during therapy. The drug should be discontinued immediately in cases of aggravated liver dysfunction or active liver disease.
3. Periodic eye examinations, including slit-lamp, funduscopy and tonometry, are recommended for patients being treated with this drug since many phenothiazines and related drugs have been shown to cause eye changes.
4. Complete urinalysis and BUN should be done on patients treated with Tegretol because of observed renal dysfunction.
Adverse Reactions Dizziness, drowsiness, unsteadiness on the feet, nausea, vomiting, aplastic anemia, transitory leukopenia, agranulocytosis, eosinophilia, leukocytosis, thrombocytopenia, purpura, abnormalities in liver function tests, cholestatic and hepatocellular jaundice, urinary frequency, acute urinary retention, oliguria with elevated blood pressure, albuminuria, glycosuria, elevated BUN, microscopic deposits in the urine, impotence, disturbances of coordination, confusion, headache, fatigue, blurred vision, transient diplopia and oculomotor disturbances, speech disturbances, abnormal involuntary movements, peripheral neuritis and paresthesias, depression with agitation, talkativeness, nystagmus, tinnitus, paralysis and other symptoms of cerebral arterial insufficiency, pruritic and erythematous rashes, urticaria, Stevens-Johnson syndrome, photosensitivity reactions, alterations in skin pigmentation, exfoliative dermatitis, alopecia, diaphoresis, recurrence of thrombophlebitis, erythema multiforme and nodosum, aggravation of disseminated lupus erythematosus, gastric distress, abdominal pain, diarrhea, constipation, anorexia, dryness of the mouth and pharynx, glossitis, stomatitis, fever, chills, adenopathy, lymphadenopathy, aching joints and muscles, leg cramps, conjunctivitis, left ventricular failure, aggravation of hypertension, hypotension, syncope and collapse, edema, aggravation of coronary artery disease and congestive heart failure. (Whether these cardiovascular effects are drug-related is not known. However, some of these complications have resulted in fatalities.) The necessity for discontinuing the drug should be dictated by the gravity and severity of the adverse reactions.
Dosage and Administration The drug should always be taken with meals, if possible.
Initial: One-half tablet (100 mg.) b.i.d. on the first day. Thereafter, the dose should be increased in one-half tablet (100 mg.) increments every 12 hours until freedom from pain is achieved. To relieve pain, between 200 mg. and 1200 mg. per 24 hours may be necessary.
Maintenance: Initial control of pain can be maintained in most patients with a dose of 400 mg. to 800 mg. daily. Maintenance doses may range between 200 mg. and 1200 mg. daily.
At least once every 3 months during treatment period attempts should be made to discontinue the drug or to reduce the dose to the minimum effective level.
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Indications: Hypertension. **Contraindications:** History of mental depression, hypersensitivity, and most cases of severe renal or hepatic diseases. **Warning:** With the administration of enteric-coated potassium supplements, which should be used only when adequate dietary supplementation is not practical, the possibility of small-bowel lesions (obstruction, hemorrhage, and perforation) should be kept in mind. Surgery for these lesions has frequently been required and deaths have occurred. Discontinue coated potassium-containing formulations immediately if abdominal pain, distention, nausea, vomiting, or gastrointestinal bleeding occur. Discontinue 1 week before electroshock therapy, and if depression or peptic ulcer occurs. **Use in pregnancy:** Regroton should be used in pregnant patients or in women of childbearing potential only when, in the judgment of a physician, its use is deemed essential to the welfare of the patients; adverse reactions (thrombocytopenia, hyperbilirubinemia, altered carbohydrate metabolism, etc.) are potential problems in the newborn. **Precautions:** Antihypertensive therapy with Regroton should always be initiated cautiously in post-sympathectomy patients and in patients receiving ganglionic blocking agents, other potent antihypertensive drugs, or curare. Reduce dosage of concomitant antihypertensive agents by at least one-half. To avoid hypotension during surgery, discontinue Regroton therapy two weeks prior to elective surgical procedures. In emergency surgery, use, if needed, anticholinergic or adrenergic drugs or other supportive measures as indicated. Because of the possibility of progression of renal damage, periodic kidney function tests are indicated. Discontinue if the BUN rises or liver dysfunction is aggravated. Hepatic coma may be precipitated. Electrolyte imbalance, sodium and/or potassium depletion may occur. If potassium depletion should occur during therapy, Regroton should be discontinued and potassium supplements given, provided the patient does not have marked oliguria. Take particular care in cirrhosis or severe ischemic heart disease and in patients receiving corticosteroids, ACTH, or digitalis. Salt restriction is not recommended. Use cautiously in patients with ulcerative colitis or gallstones (biliary colic may be precipitated). Bronchial asthma may occur in susceptible patients. **Adverse Reactions:** The drug is generally well tolerated. The most frequent side effects are nausea, gastric irritation, vomiting, diarrhea, constipation, muscle cramps, headache, dizziness and acute gout. Other potential side effects include angina pectoris, anxiety, depression, bradycardia and ectopic cardiac rhythms (especially when used with digitalis), drowsiness, dull sensorium, hyperglycemia and glycosuria, hyperuricemia, lassitude, restlessness, transient myopia, impotence or dysuria, orthostatic hypotension which may be potentiated when chlorthalidone is combined with alcohol, barbiturates or narcotics, leukopenia, aplastic anemia, skin rashes, thrombocytopenia, agranulocytosis, nasal stuffiness, increased gastric secretions, nightmare, purpura, urticaria, ecchymosis, weakness, uveitis, optic atrophy and glaucoma, and pruritus. Eruptions and/or flushing of the skin, a reversible paralysis agitans-like syndrome, blurred vision, conjunctival injection, increased susceptibility to colds, dyspnea, weight gain, decreased libido, dryness of the mouth, deafness, anorexia, and pancreatitis when epigastric pain or unexplained G.I. symptoms develop after prolonged administration. Jaundice, xanthopsia, paresthesia, photosensitization and necrotizing angiitis are possible. **Average Dosage:** One tablet daily with breakfast. **Availability:** Pink, single-scored tablets in bottles of 100 and 1000. **For details, see complete Prescribing Information.** (B)46-600-B

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PRECAUTIONS: (1) Desipramine hydrochloride should not be substituted for hospitalization when risk of suicide or homicide is considered grave. Suicidal ingestion of large doses may be fatal. (2) If serious adverse effects occur, reduce dosage or alter treatment. (3) In patients with manic-depressive illness a hypomanic state may be induced. (4) Discontinue drug as soon as possible prior to elective surgery.

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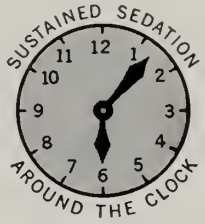
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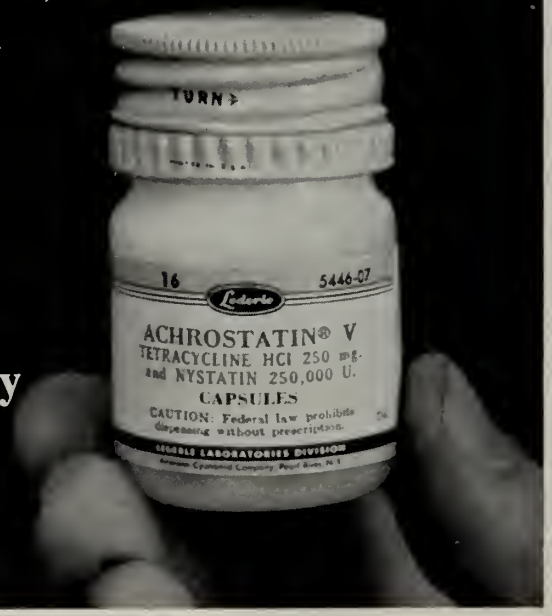
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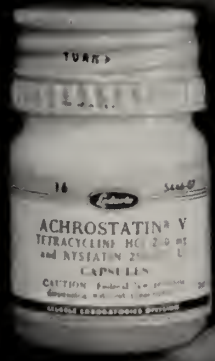
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